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A MANUAL

OF THE

PRACTICE OF MEDICINE.

DESIGNED FOR THE USE OF

STUDENTS AND THE GENERAL PRACTITIONER.

BY

HENRY C. MOIR, M. D.

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THIRD EDITION.

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NEW YORK:
J. H. VAIL & COMPANY.
1884.

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To

AMBROSE LOOMIS RANNEY, A.M., M.D.,

ADJUNCT PROFESSOR OF ANATOMY IN THE MEDICAL DEPARTMENT OF THE UNIVERSITY OF NEW YORK,

This work is respectfully dedicated, as a mark of esteem and gratitude for his many acts of kindness, by his friend and late student,

The Author.

PREFACE TO THE THIRD EDITION.

In presenting this edition to the profession, I have endeavored to keep pace with the standard literature of the day.

To this work I have added (on page 34) the causes of Hepatic Abscess, and (on page 399) a chapter on Chorea.

H. C. M.

UNION HILL, New Jersey.

PREFACE TO THE SECOND EDITION.

The success with which my exertions have been crowned in this little work, has far exceeded any reasonable expectations, and it has thereby encouraged me to bring out my second edition.

During the revision of this edition, I have endeavored to follow, as far as possible, all suggestions offered by many friends, thereby enabling me to make this work as nearly complete as possible, thus enhancing its value, both to the student and the busy practitioner.

I have especially to thank the editors of the various medical journals, for the favorable manner in which the work has been received by them, and for many valuable hints thrown out during their remarks.

The work has been enlarged; several of the diseases have been entirely re-written, and all errors, wherever detected, carefully expunged.

PREFACE.

In a small work like the present manual, nothing can possibly be given which will supply the place of exhaustive treatises. It must, of necessity, either presuppose some erudition on the part of the reader, or be elementary in character to have any special value.

This volume has been designed chiefly to aid the medical practitioner and student in refreshing their remembrance of the pathology, etiology, symptomatology, differential diagnosis, and treatment of the more important diseases.

By grouping the various causes of the more prominent *diagnostic symptoms* which are encountered by the physician, an effort has been made to impress upon the reader that no diagnosis should ever be final without a *careful study of each individual symptom;* and that, to be accurate and rapid in diagnosis, the *causes* of each and every prominent symptom of disease should be particularly memorized and constantly reviewed.

This volume, as it now appears, comprises the substance of a course given by a well-known instructor to his private students as a preparation chiefly for competitive examination, and embraces also a careful résumé of such standard works as those of Niemeyer, Roberts, Loomis, Da Costa, Bristowe, Hartshorne, and others.

To be a *skillful* diagnostician, as it seems to the author, one should first *know every disease* which can affect any tissue or organ; he

should, furthermore, have at his command the *causes in full of each and every prominent symptom* which the patient may present; and, finally, be able, by a thorough familiarity with the symptoms of each disease, *to exclude*, from the list of causes of each symptom, *such as are not sustained* by other points in the history of the patient.

By such a process of deductive reasoning, diagnosis becomes more nearly a positive science than by relying upon "keenness of perception," or "inborn intuition," which, although valuable as aids, are often worthless when the exigencies of the case demand a positiveness of diagnosis.

The arrangement of this volume is intended, as far as possible, to assist the reader to master in succession (1) the general basis on which the symptoms of disease are divided; (2) all the diseases which may be met with in each of the various organs or tissues; (3) the *more important* symptoms of disease and their etiology; and (4) the special points pertaining to the diagnosis and prognosis of individual forms of disease. This general plan of arrangement will be found carried out in the successive chapters of this volume, with an approach to completeness.

The author has appended to this volume a number of reliable and valuable prescriptions which have been obtained from prominent medical instructors and members of the profession who have fully proven their value, so as to afford the reader a pocket-memorandum of treatment of all the more important diseases; while the full details of the treatment of each disease have been incorporated throughout the text. Many points of value as to the pathology, etiology, and the diagnostic symptoms have been selected from the valuable lectures of the more prominent professors of the United States, while no work of prominence has been slighted in the preparation of this book.

The prescriptions, at the close of the manual, are arranged according to their therapeutical uses, in alphabetical order, so as to enable the practitioner to turn at once to the disease for which he seeks a recipe.

The use of italics, throughout the volume, has been adopted to make prominent such symptoms as are of special value in diagnosis.

The scope of the work will be found to be quite extensive, notwithstanding its small compass, since condensation has been the aim of the author in every page, where brevity did not demand any important omission.

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GENERAL INTRODUCTION.

GENERAL INTRODUCTION.

SYMPTOMS or SIGNS of disease comprise "any past or present circumstances afforded by the examination of a patient, or of matters concerning him, whence a conclusion may be drawn regarding the nature and seat of existing disease."

Symptoms are usually classed under one of two heads, Rational or Physical.

A RATIONAL SIGN of disease implies, by its derivation, one which is perceived by the intelligence only. It is, however, generally used in a more liberal sense, and may be said to comprise all forms of manifestations of disease of which the patient is self-conscious, or which is capable of being perceived by the patient, in case it has escaped attention.

A PHYSICAL SIGN, on the contrary, is one which, according to its derivation, must be perceived by the senses, in contra-distinction to the reasoning faculties. It is therefore, as a rule, unperceived by the patient or friends, and requires *a special cultivation of touch, sight, or hearing* before it can be accurately determined.

RATIONAL SIGNS OF DISEASE.

The rational symptoms of disease form the chief means of diagnosis, except in conditions of the respiratory organs, the heart, and the blood-vessels.

Percussion may, however, be of value in other conditions, since it assists in mapping out the situation of organs, or the outline of tumors; but the rational symptoms are, as a rule, the great and often the only guides in diagnosis, excepting in case of the organs above mentioned.

Among the more prominent of the innumerable forms of *rational symptoms* of disease may be enumerated (1) variations in the pulse and temperature, (2) pain, in all its forms and situations, (3) cough and expectoration, in all its varieties, (4) nausea and vomiting, (5) chill, (6) dyspnœa, (7) diarrhœa, (8) jaundice, (9) coma, (10) ascites, (11) hemorrhage from the different organs, (12)

aphonia, (13) constipation, (14) paralysis in all its forms, (15) abnormal conditions of urine, and (16) abnormal attitudes and physiognomy.

PHYSICAL SIGNS OF DISEASE.

Physical symptoms are of value in determining, during life, the anatomical changes taking place in tissues and organs. In order to dectect them, the following methods are employed: (1) *inspection*, (2) *palpation*, (3) *percussion*, (4) *auscultation*, (5) *mensuration*, and (6) *succussion*.

INSPECTION reveals the following points of diagnostic value: Condition of the surface; abnormalities of outline and contour (if present); deformities; abnormal growths, their character and situation; abnormal or deficient movement of parts, especially in the chest and abdomen; abnormal points of pulsation; abnormal conditions of vessels; abnormal physiognomy or attitude of some special region of the body.

PALPATION (sense of touch) reveals the following points of diagnostic value: Condition of the surface; condition of the deeper tissues; outline and character of new growths; abnormality of movement of parts, as of chest and abdomen, of tumors, the heart impulse, etc.; abnormality of transmitted impulses, as in vocal fremitus, cough impulse of hernia, etc.

PERCUSSION, by the note produced, determines the *relative solidity* of parts. Its note varies between that obtained over cavities filled with air and having thin, tense walls, called the *tympanitic* percussion note; and that obtained over fluid or solid tumors, where all air is excluded, called *flat* percussion. The percussion note is sometimes also modified by the *force of the percussion stroke*, since the flat note of solid structures is often masked by the presence of confined air between the solid body and the surface. This seldom occurs, however, in the case of fluids.

Percussion may be performed in one of two ways: directly upon the part, called "*immediate*," and indirectly through some intervening substance, as the finger, etc., called "*mediate*." In the percussion note, as in all forms of sound, the variations are confined to one of the four elements of sound, viz., its *intensity*, *pitch*, *duration*, and *quality*.

The PITCH modifies both the *intensity* and *duration* of all sounds as follows: *High pitch* is accompanied by *short duration* and *great intensity*. *Low pitch* is accompanied by *long duration* and *diminished intensity*. This fact is evidenced in every-day life by the intolerance felt towards shrill piercing notes, and the absence of such feeling when the pitch is low.

The *intensity* may be also increased, irrespective of the pitch, by the force of the blow.

The *pitch* is modified in disease by the solidity of the underlying structures; being high or flat when no air is present, and proportionately lower as the tissues approach the density of air.

The *quality* is modified in health by the character of the tissues over which percussion is made, and it varies also in certain localities with the diseased conditions of the parts. The following names have been given to percussion sounds as expressive of qualities peculiar to certain states of the body. (1) *Dullness*, if over partial consolidation; (2) *flatness*, if over complete consolidation or fluid; (3) *amphoric*, if over air in a small cavity with tense walls (this sound may be imitated by blowing into a bottle); (4) *tympanitic* if over air in a large cavity with tense walls; (5) *vesiculo-tympanitic*, as in emphysema; (6) *vesicular*, the normal percussion note of lung-tissue; (7) *wooden*, where a peculiar ring or wooden character is present; (8) *cracked-pot*, (which requires a large superficial cavity with thin tense walls, communicating with a bronchus). Firm percussion should be employed to produce this variety of percussion-note, and the patient's mouth should be kept open; (9) *auscultatory*, where a stethoscope is used simultaneously with percussion.

AUSCULTATION (sense of hearing) admits of the detection of abnormal sounds, which are often of great diagnostic value. These sounds vary with different diseases, and are of special value in diseases of the lungs, heart, and blood-vessels.

The prominent *auscultatory signs* of disease may be thus enumerated:

VARIETIES OF RESPIRATION.

Normal or "Vesicular"—is a breezy sound, like the rustling of wind through the trees.

Rude or "Broncho-vesicular"—is due to *partial consolidation* of lung—is an admixture of tubular and vesicular breathing.

Tubular or "Bronchial"—is due to *complete consolidation* of lung.

Exaggerated or "Puerile"—so called since it is normal in childhood—is produced when the lung is doing double duty.

Amphoric—occurs in cavities with *tense walls*, opening into a bronchus, and near the surface.

Cavernous—occurs in cavities with *flaccid walls*, opening into a bronchus and near the surface.

Emphysematous—where the *expiratory sound* is *prolonged* and of *low pitch.*

Phthisical—where the *expiratory sound* is *prolonged* and of *high pitch.*

Cog-wheel—where the inspiratory sound is *jerking* and *interrupted.*

Varieties of Râles.

Dry Râles.—

Sonorous—due to *partial occlusion* of a *large bronchial tube* by tumefaction of the mucous membrane, spasm of the muscular fibre, or mucus.

Sibilant—due to a similar condition of a *small bronchial tube.*

Moist Râles.—

Crepitant—due to the agglutination of the walls of an air-cell, which are subsequently separated by the entrance of air.

Mucous—due to the flapping of mucus in the bronchial tubes, or the passage of air through mucus, pus, serum, or blood.

Sub-crepitant—due to *fluid* or *mucus* in the ultimate capillary bronchi.

Gurgles—due to bubbling of air through fluid in a *cavity* in the lung, where the bronchus is below level of fluid (has a peculiar metallic, hollow sound).

Mucous-click—due to sudden and forcible passage of air through a small bronchus, whose sides are brought together by external pressure or agglutination of their walls.

Of these râles, all but one are heard with both inspiration and expiration, this one exception being the *crepitant* râle which is heard only at the end of inspiration.

AN ENUMERATION OF THE VARIOUS DISEASED CONDITIONS OF THE DIFFERENT PARTS OF THE BODY.

DISEASED CONDITIONS OF THE BODY.

DISEASES OF THE NERVOUS SYSTEM.

Diseases within the Cranial Cavity.

Inflammatory Conditions.

Acute meningitis. — Subacute meningitis.—Chronic meningitis.—Acute tubercular meningitis. — Chronic tubercular meningitis.—Cerebro-spinal meningitis, epidemic and sporadic. —Pachymeningitis, externa and interna.

Tumors.

Carcinoma.—Cholesteatoma.—Cysts.—Glioma (exists in nerve-tissue).—Gummata.—Hydatids.—Lipoma.—Myxoma.—Psammoma.—Sarcoma.—Tubercle.

Conditions of Brain Substance.

Abscess. — Atrophy.—Cerebral concussion.—Hypertrophy.—Sclerosis.—Red softening.—White softening.—Yellow softening.—Tumors, as above (except glioma).

Conditions of Vessels.

Anæmia. — Apoplexy, cerebral. — Apoplexy, meningeal.—Atheroma. — Embolism.—Fatty degeneration.—Hyperæmia, active. — Hyperæmia, passive.—Miliary aneurism.—Thrombosis.

Diseases within the Spinal Cord.

Inflammatory Conditions.

Cerebro-spinal meningitis.—Diffuse idiopathic spinal meningitis.—Localized idiopathic spinal meningitis.—Traumatic spinal meningitis.

Tumors of the Spinal Meninges.

Carcinoma.—Cysts. — Enchondroma.—Gummata.—Hydatids. —Lipoma.—Myxoma.—Sarcoma.—Spina bifida.—Tubercle.

Conditions of the Spinal Marrow.

Abscess. — Concussion. — Locomotor ataxia. — Sclerosis. —

White softening.—Red softening or myelitis.—Tumors (as above, excepting Spina bifida).

Conditions of Vessels.

Active hyperæmia.—Passive hyperæmia or "congestion."—Anæmia. — Apoplexy, meningeal or spinal. — Atheroma. — Embolism. — Fatty degeneration.—Thrombosis.

ORGANS OF DIGESTION.

Diseases of the Buccal Cavity.

Inflammatory Conditions.

Simple or catarrhal stomatitis.—Mercurial stomatitis.—Croupous stomatitis.—Aphthous sore mouth.—Diphtheritic stomatitis. — Cancrum oris. — Exudative stomatitis. — Thrush or "sprue." — Gangrenous stomatitis. — Noma or "water canker."

Mucous patches (syphilitic).

Ulcers (non-specific).

Parotitis or "mumps."

Glossitis.

Scorbutic condition.

Lead affections.

Gummata of tongue.

Surgical Diseases.

Epithelioma.—Tumors of bone.—Necrosis.—Caries.—Epulis.—Parulis.—Ranula.—Cleft-palate.—Hare-lip.

Eruptions of the different fevers.

Angina Ludovici.

Diseases of the Pharynx.

Inflammatory Conditions.

Catarrhal pharyngitis. — Croupous pharyngitis.—Follicular pharyngitis. — Diphtheritic pharyngitis. — Phlegmonous pharyngitis.—Retro-pharyngeal abscess.—Gangrene.—Angina Ludovici.—Ulcers.

Syphilitic Conditions.

Specific catarrh.—Specific ulcers.—Mucous papules.—Condylomata.—Gummata.—Cicatrices.—Constrictions and distortions of pharynx, from adhesions or cicatrizations.

Cancer of the pharynx.
Polypi of the pharynx.

Diseases of the Œsophagus.

Inflammatory Conditions.

Catarrhal œsophagitis.—Croupous œsophagitis.—Diphtheritic œsophagitis. — Pustulæ (small-pox).— Ulcers, as in chronic catarrh.—Ulcers from corrosive substances.—Ulcers from foreign bodies.

Stricture as a result of

Cicatrices.—Compression (from outside sources). — Hypertrophy of its walls.—New growths in the œsophagus.

Dilatation of the Œsophagus.

Partial dilatation, if circumscribed.—Total dilatation if affecting the entire length.

New Growths.

Carcinoma.—Fibroid tumors.—Gummata (rare). — Tubercle (rare).

Perforation or Rupture.

From abscess of bronchial glands.—From aneurism.—From cancer.—From caries of the vertebræ.—From corrosion.—From ulcers.

Nervous Conditions.

Globus hystericus (spasm).—Hyperæsthesia.—Paralysis.

Diseases of the Stomach.

Inflammatory Conditions.

Acute catarrhal gastritis.—Chronic catarrhal gastritis.—Subacute catarrhal gastritis.—Croupous gastritis.—Diphtheritic gastritis.—Phlegmonous gastritis.—Toxic gastritis.—Ulcers, acute or perforating, and chronic.—Cancer, scirrhus, medullary, and colloid.

Nervous Conditions.

Dyspepsia.—Spasm or "gastralgia."

Unclassified Conditions.

Dilatation of the stomach.—Rupture of the stomach.—Stricture of the stomach.

Hemorrhage dependent upon

Ulcer.—Cancer.—Thrombosis of portal vein.—Cirrhosis of the

liver.—Acute yellow atrophy of the liver.—Enlargement of gall-ducts.—Pulmonary obstruction.—Tricuspid regurgitation.—Hemorrhagic diathesis.—Rupture of aneurism and varices.—Traumatism.—Improper living (as abstinence from meat or vegetables).

Blood Conditions.

Yellow fever.—Malaria.—Scurvy.—Purpura.

Ulcers.

Acute or perforating.—Chronic catarrhal.—Phlegmonous.—Scrofulous.—Syphilitic.—Diphtheritic.—Cancerous.—Variolous.—Corrosive (due to poisons).—Typhus.—Typhoid.—Follicular.

Diseases of the Intestinal Canal.

Inflammatory Conditions.

Acute and chronic catarrhal enteritis.—Gastro-duodenitis (cholera morbus).—Perforating duodenal ulcers (as occur in burns).—Peri-typhlitis.—Proctitis and peri-proctitis.—Typhoid conditions.—Dysentery, sporadic and epidemic.—Cholera morbus.—Asiatic cholera.

Tumors.

Vascular.—Carcinoma.—Tubercle.—Fatty tumors (lipoma).—Scrofulous enlargements of the mesenteric glands.

Conditions Dependent upon the Vessels.

Intestinal hemorrhage.—Vascular dilatations.

Conditions impairing the free action of the Bowels.

Contractures, by adhesions or bands of lymph.—Rupture or perforation.—Foreign bodies.—Volvulus.—Gall-stones.—Intussusception.—Peritoneal bands.—Hernia.—Worms.—Strangulation.—Pressure of tumors and cancer.—Impaction of fæces.

Unclassified Conditions.

Worms.—Tympanites or wind colic.—Intestinal colic.—Lead colic.

Diseases of the Liver.

Inflammatory Conditions.

Affecting the capsule of the organ (peri-hepatitis).—Affecting the parenchyma of the liver (diffuse hepatitis).—Affecting the connective tissue (cirrhosis).—Circumscribed suppurative inflammation (abscess).—Ascites.—Jaundice.

Degenerations.

Amyloid or waxy liver.—Fatty liver.—Acute yellow atrophy.—Chronic atrophy from pressure.

Abnormal Conditions of the Vessels.

Active hyperæmia.—Passive hyperæmia (nutmeg liver).—Hepatic apoplexy.—Hepatic infarction.—Portal thrombosis.—Suppurative portal phlebitis.—Embolism of hepatic artery.

Hepatic Tumors.

Cancerous tumor.—Tubercle.—Cysts, simple and hydatid.—Carcinoma.—Gummata.—Enlarged gall-bladder.

Conditions of Bile Ducts.

Gall-stones.—Catarrhal obstruction.—Croupous obstruction.—Foreign bodies in the duct.—Tubercle.—Cancer.

ORGANS OF RESPIRATION.

Diseases of the Larynx.

Inflammatory Conditions.

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Ulcers.

Catarrhal.—Typhus.—Variolus.—Syphilitic.—Tubercular.

New Growths.

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Nervous Conditions.

Laryngeal spasm (false croup).—Paralysis of muscles of larynx.

Diseases of the Bronchi.

Inflammatory Conditions.

Acute catarrhal bronchitis.—Chronic catarrhal bronchitis.—Capillary catarrhal bronchitis.—Croupous bronchitis.—Diphtheritic bronchitis.—Bronchial dilatation.

Nervous Conditions.

Asthma.—Whooping cough.

Surgical Conditions.

Foreign bodies.—Occlusion by pressure, or from tumors.

Diseases of the Lungs.

Inflammatory Conditions.

Croupous or lobar pneumonia.—Catarrhal or lobular pneumonia.—Chronic, or interstitial pneumonia.—Pulmonary abscess.—*Pulmonary gangrene.—*Pulmonary œdema.

Conditions dependent upon the vessels of the Lung.

Pulmonary apoplexy. — Pulmonary infarction.—Pulmonary œdema. —Pulmonary gangrene. —Active hyperæmia.—Passive hyperæmia.—Compensatory hyperæmia.—Brown induration. —Splenization.—Hypostatic congestion.

Conditions dependent upon an altered state of the Air-cells.

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Inflammatory Conditions.

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Hæmo-thorax.

Hydro-pneumo-thorax.

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Diseases of the Heart and Pericardium.

Inflammatory Conditions.

Acute endocarditis.—Chronic endocarditis.—Ulcerative endocarditis.—Myocarditis. —Pericarditis, with serous effusion.— Pericarditis, with pus exudation.—Pericardial adhesion.

Abnormal Valvular Conditions.

Aortic obstruction. — Aortic insufficiency. — Pulmonary

* Pulmonary œdema and gangrene are not always of inflammatory origin; hence enumerated under two headings.

obstruction and insufficiency. — Mitral obstruction and insufficiency. — Tricuspid obstruction and insufficiency. — Vegetations on the valves.—Rupture of chordæ tendineæ.—Hypertrophy, 3 varieties, *simple, eccentric,* and *concentric.*—Rupture of the heart.

Unclassified Conditions of the Heart.

Fatty degeneration of the heart.—Amyloid degeneration of the heart.—Angina pectoris.—Cardiac neuralgia.—Heart clot, or cardiac thrombosis.—Cardiac polypus.—Cardiac dilatation, 3 varieties, *simple, hypertrophous,* and *atrophic.*

Conditions of the Pericardium.

Basedow's disease. — Pneumo-pericardium. — Hæmo-pericardium.—Cancer of pericardium.—Aneurism in pericardial sac.—Tubercle of pericardium.

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Renal Tumors.

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In the Cavity of the Kidney.

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Addison's disease.

Diseases of the Bladder.

Inflammatory Conditions.

Acute catarrhal cystitis.—Chronic catarrhal cystitis.—Ulcer-

ation. — * Paralysis. — Croupous cystitis.— Diphtheritic cystitis.

Tumors.

Cancer.—Tuberculous.—Vegetations.

Nervous Conditions.

Atony.—Spasm.—Paralysis.—Neuralgia.

Conditions affecting the cavity of the Organ.

Dilatation.—Rupture.—Retention.—Incontinence.—Overflow. —Calculus in the bladder.—Hemorrhage.

A CLASSIFICATION OF THE ABNORMAL STATES OF THE BLOOD.

Changes in its Corpuscular Elements.

Increase of red globules (plethora).—Increase of white globules (leucocythemia).—Decrease of red globules (anæmia).—Decrease of red and white globules (chlorosis).—Disorganization of globules (fevers).

Changes in its Inorganic Elements.

Increase in chloride salts.—Increase in alkaline salts (some nervous diseases).—Decrease in chloride salts.—Decrease in alkaline salts (scurvy).

Changes in its Organic Elements.

Incresase in fibrin (starvation, inflammation).—Increase in albumen (inflammation).—Decrease in fibrin (all the fevers). — Decrease in albumen (all the fevers).—Increase in fat (chylæmia).—Decrease in fat (tuberculosis) (?).

Changes Dependent upon Mal-assimilation.

Excess of lactic acid (rheumatism).—Excess of uric acid (gout).—Excess of oxalic acid (oxaluria).—Excess of sugar (diabetes).

Changes due to Poisons Developed within the Body.

Uræmia.—Ammonæmia. — Cholestræmia.—Cyanosis.—Intestinal secretion (fetid odor to breath).

Changes due to Poisons Developed outside the Body.

Glanders.—Anthrax.—Hydrophobia.—Snake-bite.—Syphilis. —All the fevers.—Dysentery. — Cholera. —Cerebro-spinal meningitis (?).—Pyæmia.—Septacemia.—Fevers.

* May also be dependent on many other causes.

PROMINENT SYMPTOMS OF DISEASE, POSSESSING A SPECIAL DIAGNOSTIC IMPORTANCE, AND THEIR VALUE.

PROMINENT SYMPTOMS OF DISEASE, POSSESSING A SPECIAL DIAGNOSTIC VALUE.

VARIETIES OF PULSE.

Modifications in Volume.

Full pulse.—Small pulse.—Thready pulse.—Gaseous pulse.

Modifications in Rhythm.

Regular, where the rhythm is perfect.—Irregular, where the number of beats per minute or fraction of a minute is not uniform.—Intermitting, where the beats cannot be perceived at intervals.—Delayed, where the radial pulse and the apex beat are not synchronous with each other.

Modifications in Force.

Weak pulse.—Strong pulse.—Suppressed pulse.—Jerking pulse.—Compressible pulse.—Incompressible pulse.—Wiry pulse.—Shot pulse (in aortic regurgitation).

Modifications shown by the Sphygmograph.

Dicrotic, where the *aortic wave* is prominent and the aortic notch *approximates* toward the *respiratory line.*—Fully Dicrotic, where the *aortic wave* is prominent and the aortic notch *on a level* with the respiratory line.—Hyper-dicrotic, where the aortic notch falls below the respiratory line.

The depth of the *aortic notch* shows the tension or emptiness of the vessels. The less the depth the greater the tension. The greater the depth the greater the emptiness of the vessels.*

CAUSES OF EPIGASTRIC PULSATION.

Aneurism of cæliac axis.
Aneurism of abdominal aorta.

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* A state of *great tension* after scarlet fever is indicative of a developing kidney complication.

Tumors lying upon the abdominal aorta.
Displacement of the heart toward the right side.
Pulsation in the hepatic veins and inferior vena cava (due to tricuspid regurgitation).
Transmission of the heart impulse through the left lobe of the liver.
A relaxed condition of the abdominal aorta, or its loose attachment to the vertebræ.
Hypertrophy of right auricle (as in emphysema, tricuspid regurgitation, etc.).

CAUSES OF DISPLACEMENT OF APEX BEAT OF HEART.

Downward.

Emphysema.—Mediastinal tumors.—Cancer of lung or upper part of pericardium.—Cardiac dilatation.—Hypertrophy of right and left ventricles of heart.

Upward.

Dilatation of right ventricle of heart (?).—Distended stomach. Enlarged left lobe of liver.—Enlarged spleen.—Tympanites.—Abdominal tumors (if very large).—Ascites (if extensive).

Laterally.

Fluid in pleural cavities.—Pleuritic adhesions.—Pleurisy.—Emphysema.—Mediastinal tumors.—Abdominal tumors (if the development is most extensive on the right side).

CAUSES OF ABDOMINAL PAIN.

Conditions of Stomach.

Gastritis (all varieties).—Ulcers of stomach.—Cancer of stomach.—Distention of stomach.—Rupture of stomach.—Gastrodynia.

Conditions of Intestines.

Enteritis (all varieties).—Dysentery (all varieties).—Impaction of fæces.—Stoppage of bowel, and its causes.—Tympanites.—Wind colic.—Lead colic.—Typhoid conditions.—Tuberculous ulcers.—Perforation.—Typhlitis.—Peri-typhlitis.—Peri-proctitis.—Cancer.

Conditions of Liver.

Hepatic colic (gall-stones).—Cirrhosis.—Hepatitis.—Peri-hepa-

titis.—Abscess of liver.—Cancer of liver.—Hydatids of liver.—Tumors of liver.—Pylo-phlebitis.

Conditions of Kidney.

Renal colic (gravel).—Renal abscess.—Pyelitis.—Pyo-nephrosis.—Hydatids.—Cancer.—Peri-nephritic abscess.—Addison's disease.

Conditions of Spleen.

Malarial enlargement.—Leucocythemia (by pressure on other organs).

Conditions of Peritoneum.

Localized peritonitis.—Tuberculous peritonitis.—Acute peritonitis.—Cancer of omentum.

Conditions of Ovary.

Ovarian tumors.—Prostitutes' colic (ovaritis).

Conditions of Uterus.

Metritis. — Endo-metritis.— Cancer. — Rupture.—Polypus.—Tubal pregnancy.—Flexions.

Conditions of Bladder.

Cystitis (all forms).—Retention of urine.—Rupture of bladder.—Spasm of bladder.—Cancer of bladder.—Ulceration of bladder.—Calculus.

Conditions of Vessels.

Aneurism.—Hæmatocele.

CAUSES OF PAIN IN THE THORAX.

Affecting Respiratory Organs.

In the Lung.

Bronchitis (acute).—Pneumonia.—Cancer.—Abscess.—Infarction.—Phthisis.—Foreign bodies in bronchi.

In the Pleura.

Pleurisy (all varieties).—Pneumo-thorax.—Hæmo-thorax.—Cancer.

Located in Mediastinæ.

Mediastinal tumors.—Diaphragmatic hernia.

Affecting Heart and Coverings.

In the Heart and Pericardium.

Pericarditis (all varieties). — Pneumo-pericardium.—Hæmo-

pericardium.—Cancer of pericardium.—Myocarditis.—Ulcerative endocarditis.—Angina pectoris.—Cardiac neuralgia.

AFFECTING WALLS OF THORAX.

Surgical Conditions.

Contusions.—Traumatisms.—Fracture of ribs.—Fracture of vertebræ (dorsal).—Dislocation of vertebræ (dorsal).

Diseased Conditions.

Intercostal neuralgia.—Muscular rheumatism.—Abscess in soft tissues over the thorax.—Cancer of soft tissues.—Cancer of thoracic walls.—Tumors of thoracic walls.—Stricture of œsophagus.

Abdominal Causes.

Dyspepsia.—Abdominal pressure upward.

CAUSES OF VOMITING.

LOCAL.

Gastritis (all varieties).—Gastric cancer.—Gastric ulcer.—Dyspepsia.—Stricture of stomach.—Cirrhosis of liver (from passive hyperæmia of the stomach).—Cholera (from desquamation of the epithelium).—Cholera morbus.—Impaction of fæces.—Peritonitis.—Strangulated hernia.—Volvulus.—Intussusception.—Worms in stomach or intestines.—Mechanical irritants (as mustard, emetics, poisons, etc.).

REFLEX.

Diseases of uterus.—Diseases of ovaries.—Diseases of kidney.—Diseases of spleen.—Diseases of liver.—Diseases of bladder.—Diseases of testicle.—Diseases of brain.—Diseases of spinal cord.—Pregnancy.—Irritation of palate or fauces.—Irritation of olfactory nerve (by odors).—Severe pain (from any cause).

BLOOD.

Poisons of a Medicinal Nature.

Emetics.—Narcotics.—Cardiac sedatives.—Anæsthetics.

Poisons from the Body.

Uræmia.—Cholæmia.—Ammonæmia.—Uric acid.—Cholestræmia.

Malarial poisoning.

All the fevers.
Hemorrhage (if profuse).
Anæmia.
Chlorosis.

CAUSES OF HEADACHE.

Cranial Causes.

Anæmia of cerebral capillaries.—Congestion of cerebral capillaries.—Thrombosis of cerebral capillaries.—Embolus of cerebral capillaries.—Cerebral apoplexy.

Inflammatory Diseases.

Meningitis (9 varieties—see page 9). — Cerebritis. — Cerebral abscess.—Caries and necrosis.

Tumors of meninges and of brain substance (see page 9).
Cerebral concussion.

Reflex Causes.

Abnormal states of stomach.—Abnormal states of uterus.—Pregnancy.—Constipation.—Excessive venery.

Blood Causes.

Poisons.

All the fevers.—Malaria.—Diphtheria.—Syphilis.—Uræmia.—Cholæmia.—Cholestræmia.—Pyæmia.—Septicæmia.—Medicinal agents.—Alcohol.

Anæmia.
Chlorosis.
Scurvy.
Purpura.

CAUSES OF CHILL.

General Axiom.

Any disease in which a *marked* and *sudden rise* in temperature occurs, may be ushered in with or accompanied by a chill.

1. All INFLAMMATORY DISEASES of any of the various organs or structures of the body, if severe, may *begin with a chill.*
2. *Blood Poisons.*

   Pyæmia.—Septicæmia.—Puerperal fever.—Malaria.—All the fevers.—Erysipelas.—Surgical fever.
3. *Nervous Causes.*

   Sudden shock.—Nervous exhaustion.—Mental emotion.

CAUSES OF DYSPNŒA.

Is due to some mechanical interference to the free entrance of air to the lung.

Causes above the Larynx.

May result from pressure on facial nerve.--May result from obstruction in nares.—Suppurative tonsillitis.—Retro-pharyngeal abscess. -- Cancer of mouth or pharynx.--Tumors of mouth or pharynx.--Foreign bodies in pharynx.

Causes in the Larynx.

Laryngitis (5 forms—see page 13).—Œdema glottidis.--Laryngismus stridulus.—Laryngeal tumors.--Laryngeal paralysis.—Pressure upon the larynx.—Foreign bodies in larynx.

Causes in Bronchi.

Bronchitis (its various forms).--Asthma.--Foreign bodies in the bronchi.--Hemorrhage from a bronchus.—Pressure on a bronchus.—Bronchial dilatation.

Conditions of the Lungs.

Emphysema. -- Pneumonia. -- Phthisis. — Œdema.-- Congestion. -- Infarction. -- Apoplexy.--Abscess.— Cancer. — Gangrene.--Atelectasis.--Compression of the lung.

Conditions of the Pleura.

Pleurisy, acute. -- Pleurisy, subacute. -- Pleurisy, chronic (empyema).--Pleuritic adhesions.—Hydro-thorax.--Pneumo-thorax.—Hydro-pneumo-thorax.—Hæmo-thorax.--Cancer.

Conditions of the Heart.

Pressure on the heart.--Enfeebled heart's action.—Mitral disease.—Rupture of valves.—Angina pectoris.--Cardiac dilatations.—Fatty heart.--Myocarditis.--Endocarditis (ulcerative form chiefly).—Greatly accelerated heart's action.

Conditions of the Pericardium.

Pericarditis with effusions.--Pericardial adhesions.—Pneumo-pericardium. — Hydro-pericardium. -- Hæmo-pericardium.—Cancer of pericardium.—Pus in pericardium.

Conditions of the large Vessels.

Aneurism of arch (by pressure on lung).—Aneurism of pericardial sac (by pressing on heart).--Air in veins.

Conditions of Air Respired.

Deficiency of oxygen.—Too high altitudes.—Deleterious substances and impurities in the air.

Conditions of Blood.

Anæmia.—Chlorosis.—Poisons.

Conditions of the Nervous System.

Diseases of the brain.—Diseases of upper part of the spinal cord. —Injury to or pressure upon the following nerves: Pneumogastric, Phrenic, Spinal Accessory, Laryngeal, Cardiac.—Exhaustion.—Tetanus.—Hydrophobia.

Conditions affecting Parietes or Muscles of Chest.

Spasm of chest muscles.—All *painful affections* of structures external to chest.—Paralysis of respiratory muscles.—Wounds or contusions of soft tissues.—Ossified cartilages.—Fracture of ribs.—Dislocation or fracture of spine (in dorsal region).

Conditions affecting the movements of the Diaphragm.

Enlarged organs, including pregnancy.—Tympanites.—Ascites.—Peritonitis (from the pain).—Tumors.

CAUSES OF JAUNDICE.

Hepatogenous (Obstructive).

Affecting the common Bile-duct.

Congenital defect of the valve in the duct.—Extension of catarrhal inflammation from the intestines.—Plugging of duct by calculus.—Plugging of duct by hydatids.—Plugging of duct by mucus.—Plugging of duct by foreign bodies.—Plugging of duct by worms.—Stricture of the duct.—Pressure on duct by abdominal tumors.—Pressure on duct by enlarged lymphatics.

Affecting the Radicals of the Bile-ducts.

Cancer of the liver.—Hydatid of the liver.—Abscess of the liver.—Cirrhosis of the liver.—Gummata.—Tuberculosis.—Hyperæmia.—Hyperplastic inflammation of the capsule of Glisson, in the transverse fissure of the liver.

Hæmatogenous (Non-obstructive).

Blood Poisons.

Malaria.—Antimony.—Phosphorous.—Snake bites.—Pyæmia. —Septacemia.—Anæsthetics.—Fevers.

Emptiness of blood-vessels of the liver (favoring osmosis).
Hypersecretion of bile.
Acute yellow atrophy.
Fevers.
Pneumonia.
Mental emotions.
Secondary to the hepatogenous variety.

CAUSES OF COMA.

Cranial Causes.

By producing Pressure or Injury.

Bony tumors.—Suppuration, from caries or necrosis.—Depressed fracture. — Meningitis (9 varieties — see page 9) due to pressure from the exudation. — Meningeal hemorrhage. — Meningeal tumors (see page 9).—Abscess of brain.—Sclerosis of brain.—Apoplexy.—Tumors of the brain (see page 9).

Affecting the Generative Power of Nerve Centres.

Cerebral softenings, red, white, and yellow.—Cerebral concussion.—Cerebral anæmia.—Thrombosis.—Embolism.

Nervous Causes.

Epilepsy. — Hysteria.—Catalepsy. — Syncope (through the sympathetic).—Reflex coma (especially in children).

Blood Poisons.

All the fevers.—Malaria. — Diphtheria.—Uræmia.— Pyæmia. —Septicæmia.—Alcoholism.—Cholestræmia.—Ammonæmia. —Narcotics, such as chloral, opium, etc.—Cholæmia.—Anæsthetics.

CAUSES OF ASCITES.

In the Liver.

Pressure on portal vein from abdominal tumors.—Atrophy of liver.—Cirrhosis of liver (from pressure on portal vein).—Cancer.—Waxy liver.—Enlarged lymphatic glands in transverse fissure of the liver.—Hydatids.—Abscess.—Tubercle.—Gummata.—Portal thrombosis.

In the Heart.

Triscupid regurgitation (by causing imperfect venous return to the right auricle).

In the Lungs.

Emphysema.—Cancer.—Fluid in pleural sacs.—Mediastinal tumors (by causing insufficiency of tricuspid valve).

In the Kidneys.

Chronic Bright's (by producing a general hydræmia).

Peritoneal Causes.

Cancer of peritoneum.—Tubercular disease of peritoneum.—Chronic inflammation of peritoneum.

Blood Causes.

Anæmia.—Hydræmia.—Chlorosis.—Purpura.—Scurvy.

CAUSES OF HEMORRHAGE FROM THE DIGESTIVE TRACT.

From the Stomach (Hæmatemesis).

Dependent on Stomach.

Acute catarrhal gastritis. — Toxic gastritis.—Phlegmonous gastritis.—Gastric ulcer.—Gastric cancer.

Dependent on Hepatic Changes.

Cirrhosis of liver (by obstructing portal circulation).—Acute yellow atrophy.—Portal thrombosis.—Pressure on portal vein.

Dependent on the Vessels.

Traumatism.—Hemorrhagic diathesis (usually due to an abnormality of the coats of the blood-vessels).—Pulmonary obstruction (by producing tricuspid regurgitation).

Dependent on the Blood.

Malaria.—Scurvy.—Purpura.—Yellow fever.

Vicarious Causes.

Hemorrhoids.—Menstruation.

Intestinal Hemorrhage.

Dysentery.—Typhoid fever.—Yellow fever.—Malarial poisoning.—Tuberculous ulceration. — Cancer.—Scurvy. — Purpura — Traumatism.—Obstruction of portal vein.

CAUSES OF HÆMOPTYSIS.

Those Situated above the Lung.

Diseases of larynx.—Diseases of trachea.—Diseases of bronchi. (Such as Congestion, Inflammation, Ulceration, Cancer, etc.)

In the Lung.

Mechanical hyperæmia (from too high altitudes, and the inhalation of irritants).—Traumatism.—Pulmonary congestion.—Pneumonia, acute.—Pneumonia, chronic.—Abscess.—Gangrene.—Apoplexy.— Phthisis.—Cancer.—Weak capillaries.—Aneurism of pulmonary capillaries.—Pleuritic adhesions (causing interference with the lung tissue).

Mediastinal Causes.

Tumors, pressing on the pulmonary vessels.

Circulatory Causes.

Aneurism of arch of aorta (breaking into a bronchus).—Mitral diseases.—Hypertrophy of right ventricle.—Dilatation of left ventricle.—Diseases of pulmonary vessels (as aneurism of pulmonary artery).—Aneurism of the arteria innominata, the carotid, or subclavian arteries opening into the air-passages.

Nervous Causes.

Vicarious menstruation.

Blood Causes.

Hemorrhagic diathesis.—Scurvy.—Purpura.

CAUSES OF APHONIA.

Inflammatory.

Catarrhal laryngitis (acute and chronic).—Tuberculous laryngitis.— Syphilitic laryngitis.— Croupous laryngitis. — Diphtheritic laryngitis.

New Growths.

Cancer. — Polypi.—Vegetations.

Mechanical Causes.

Hemorrhage of vocal cords.—Paralysis of muscles of the larynx —Thickening of cords.—Spasm of cords.—Rupture of cords. Œdema of the larynx.—Foreign bodies in the larynx.—External pressure upon the larynx.

Nervous Causes.

Injury to the brain in the region of origin of spinal accessory nerve.—Injury to the trunks of the pneumo-gastric or spinal accessory nerves.—Hysteria.—Mental excitement (from fright, anger, etc.).—Diphtheria.—Shock.

CAUSES OF HEMIPLEGIA.

Cranial.

Affecting the Bones.

Depressed fracture.—Necrosis.—Caries.—Exostosis.—Cancer.

Affecting the Meninges.

Meningitis (9 varieties [see page 9]).—Tumors (Cancer, Tubercle, Cholesteatoma, Gummata, Myxoma, Lipoma, Psammoma, Hydatids, Cysts).—Meningeal hemorrhage.—Abscess (between dura mater and the bone).

Affecting the Brain Substance.

Cerebral softenings (red, white and yellow).—Cerebral tumors (see above and add "Glioma").—Sclerosis.—Abscess.

Affecting the Vessels.

Thrombosis.—Embolism.—Apoplexy.

Spinal.

(*Involving only the lateral half of spinal cord.*)

Affecting the Bones.

Same as those among the cranial causes, with *dislocation* added.

Affecting the Meninges.

Spinal meningitis.—Cerebro-spinal meningitis.—Meningeal hemorrhage.—Tumors (same as those under cranial causes).

Affecting the Substance of Spinal Cord.

White softening.—Red softening or myelitis.—Tumors (same as those under cranial causes).—Sclerosis of the cord.—Abscess of the cord.—Congestion of the cord.—Apoplexy.

Functional.

Diphtheria.—Epilepsy.—Hysteria.—Chorea.—Poisons.

CAUSES OF PARAPLEGIA.

Spinal.

Same as spinal causes of hemiplegia (provided both lateral halves of the cord are involved).

Functional.

Anæmia.—Rheumatism.—Syphilis.—Hysteria.—Diphtheria.

REFLEX.

Phymosis.—Diseases of bladder.—Diseases of uterus.—Diseases of intestinal canal.—Diseases of ovary.—Diseases of clitoris.—Diseases of kidney.

CAUSES OF SUPPRESSION OF URINE.

Acute active congestion of kidney.
Blood-poison of yellow fever.
Blood-poison of scarlet fever.
Blood-poison of small-pox.
Suppurative interstitial nephritis (following injury to or operations on the urinary tract).
Any *inflammatory diseases* may cause it.

CAUSES OF RETENTION OF URINE.

Dependent on the Urethra.

Organic urethral stricture.—Hypertrophy of prostate.—Suppuration of prostate.—Pressure on urethra from perineal abscess.—Pressure on urethra from perineal tumors.—Pressure on urethra from fracture of pelvic bones.—Rupture of urethra.—Calculus in urethra.—Spasm of urethral muscular fibres.—Congestion of urethral mucous membrane.—Fracture of penis.—Cancer of penis.—Polypus of the urethra.

Dependent on the Bladder.

Paralysis of the bladder.—Atony from over-distention or shock.—Cancer at neck of the bladder.—Vegetations at neck of the bladder.—Stone in cavity of the bladder.—Rupture of the bladder.—Pressure from tumors.

CAUSES OF BLOOD IN URINE.

LOCAL.

Dependent on the Kidney.

Hyperæmia.—Cancer.—Pyelitis.—Infarction.—Stone in pelvis.—Abscess.—Traumatism.—Parasites.

Dependent on the Ureter.

Cancer.—Traumatism.—Stricture.—Ulceration.—Impaction of calculus.

Dependent on the Bladder.

Cystitis. — Over-distention.— Cancer.—Stone in bladder. — Vegetations.—Rupture.—Ulceration.—Traumatism.

Dependent on the Urethra.

Gonorrhœa —Chordee.—Chancre or chancroid.—Cancer.—Impaction of a calculus.—Rupture of urethra.—Rupture of a prostatic abscess.—Fracture of penis.—Traumatism.

BLOOD-CAUSES.

All of the Fevers and Malarial Conditions.

Poisons.

Turpentine.—Copaiba.—Cantharides.—Cubebs.—Alcohol.

Scurvy.

Purpura.

VICARIOUS CAUSES.

In connection with amenorrhœa.

In connection with hemorrhoids.

CAUSES OF PUS IN THE URINE.

Dependent on the Kidney.

Pyelitis.—Cancer of kidney.—Metastatic abcess of kidney.—Stone in the pelvis of kidney.—Perforation of peri-nephritic abscess.

Dependent on the Bladder.

Cystitis in all its forms.—Stone in the bladder.—Cancer of the bladder.

Dependent on the Urethra.

Cancer of the penis.—Prostatic abscess.—Ulceration.—Gonorrhœa.—Urethritis.

Dependent on the Adjacent Viscera.

Escape of pus from other organs into the genito-urinary tract.

CAUSES OF INSTANTANEOUS DEATH.

In the Heart.

Aortic regurgitation.—Heart-clot.—Rupture of the heart.—Angina pectoris.—Rupture of aneurism into the pericardial sac.—Fatty heart.

Dependent on Blood-vessels.

Apoplexy.—Air in veins.

Unclassified Conditions.

Concussion of *solar plexus.*—Perforation of some *abdominal viscera* (from shock [?]).—Sudden injury to *brain* or *medulla oblongata* (as occurs in apoplexy).

TYPES OF EXPECTORATION.

Muco-purulent.

Occurs in phthisis—and is present in all forms of bronchitis, except croupous.—Appears after attacks of asthma.—Accompanies emphysema, as a rule, since it is usually complicated by bronchitis.

Pearls of Tenacious Mucus.

Are frequently detected in emphysema.

Pneumonic Sputa.

Very tenacious at all times; at first, scanty and whitish; later on, pink or brick-dust in color; in bad cases, resembles "prune juice."

Profuse-Watery.

Occurs in pulmonary œdema (prominently).

Gangrenous Sputa.

Characterized by a putrid odor, and the presence of gangrenous shreds of lung-tissue (present in gangrene of the lung and in phthisis).

Currant Juice.

Occurs in cancer of the lung.

Purulent.

May occur in abscess of lung; or in empyema, when the pleural cavity communicates with a bronchus.—Occurs also in abscess of liver, by perforation of diaphragm and pleura.

Fibrinous Casts.

Present, in shreds or patches, in true membranous croup. Present in croupous bronchitis (when they resemble a tree in form).

Sputa, with Small Blood-Clots.

Is usually characteristic of pulmonary infarction.

Sputa, Streaked with Blood.

Present, as a rule, in tuberculous phthisis during the stage of deposit.—May exist also in a severe type of bronchitis and inflammatory sore throat.

Blood.

Occurs in aneurism; pulmonary apoplexy; bronchial hemorrhage.

TYPES OF DIARRHŒA.

Symptomatic.

Typhoid fever.—Dysentery.—Cholera.—Exhaustion.—Malarial conditions.—Fatty diarrhœa.—Typhus (in later stages).—Tuberculosis (in later stages).

Irritative.

From indigested food, or any irritant (as cathartics, etc.).

Sympathetic.

In connection with worms.—In connection with dentition.—In connection with menstruation.—In connection with nervous shock.—In connection with fright.—In connection with mental exercise.

Eliminative.

An evidence of nature's attempt to eliminate, through the intestinal tract, some accumulated poison, or an excess of some salt absorbed, as in the case of saline cathartics.

CAUSES OF ALBUMINOUS URINE.

Mollities Ossium.—Excessive use of albuminous food.—Irritation of the renal nerves.—Irritation of the floor of the fourth ventricle.—Inhalation of irritants (as arsenuretted hydrogen and carbonic acid).—Dyspepsia.—Acute or Chronic Bright's Disease of the Kidneys (due to structural changes in those organs).—Pregnancy and the puerperal state (due to obstruction of the renal veins from pressure or an excess of excrementitious substances in the blood to be eliminated by the kidney).—The various fevers, as typhoid fever, scarlet fever, measles, yellow fever, small pox, cholera, ague, diphtheria, (due to pyrexia and the operation of a specific poison indicating serious structural changes in the kid-

neys).—Certain inflammatory diseases, such as articular rheumatism, peritonitis, pneumonia, traumatic fever, etc :—Obstruction to the circulation of the blood, as occurs in emphysema, valvular heart lesions, abdominal tumors, cirrhosis of the liver, atheroma, etc :—An hydræmic and disordered state of the blood and atony of the tissues, as in purpura, scurvy, pyæmia, hospital gangerene, also hæmatinuria ; and finally saturnine intoxication.

CAUSES OF CHYLOUS URINE.

Long residence in tropical climates.—A fall or shock to the nervous system.—Excessive mental or bodily work.—Cessation of lactation.—Tuberculosis of the lungs.—Fatty stage of parenchymatous nephritis.—Some derangement of the process of assimilation causing an elimination of food elements by the kidneys.—Some imperfectly understood derangements of the submucous tissues of the urinary passages or bladder.—Hypertrophy of the lymphatic or lacteal vessels, or the inguinal glands (which become enlarged and tumified and ultimately discharge their contents into the urinary passages.—It may possibly be caused by minute parasites which infest the urinary passages (?).

Characteristics of Chylous Urine :

(1st.), a whitish or milk color.—(2), *more or less perfect spontaneous solidification* of the urine on standing, which in marked cases may allow the urine to be turned out like a jelly from one receptacle to another.—(3), a strongly albuminous reaction to heat and nitric acid, and (4), the presence of fat globules in a condition resembling that of chyle when examined by the microscope.

CAUSES OF HEPATIC ABSCESS.

Varieties.—Acute, chronic, metastatic or pyæmic, and inflammatory. Wounds and contusions; impaction in the gall-ducts of angular substances; ulcerations or gangrenous affections of the abdominal organs; embolus of the branches of the portal vein or some injurious irritating matter, carried to the liver through the portal vein, or some other vein as the hemorrhoidal ; inflammation of the parenchyma (Glisson's capsule) from injuries, suppuration, thromboses and inflammation of the veins ; inflammation and suppuration of the gall-bladder or gall-ducts; exposure to vicissitudes of temperature (especially in hot countries); malarious poisons: cold; nervous depressions; pregnancy; drunkenness.

DISEASES OF THE RESPIRATORY ORGANS.

DISEASES OF THE LARYNX.

These may be *primary*, when the larynx is first affected, and of a purely local type; or *secondary*, when the disease occurs as a complication.

VARIETIES.

Catarrhal laryngitis, acute and chronic.
Œdema glottidis, or inflammation of the submucous tissue of the larynx.
Croupous laryngitis, or "membranous croup."
Ulcers—which may be catarrhal, typhus, variolus, tubercular, or syphilitic.
Nervous affections—spasm and paralysis.
New formations—polypi, cancer, tubercle, ossification of cartilages, etc.

ACUTE CATARRHAL LARYNGITIS.

DEFINITION.

Is an inflammation of the mucous membrane of the larynx, of either a partial or general character, resulting in catarrhal products.

MORBID ANATOMY.

The mucous membrane of the larynx will be found to be red, swollen, and softened, and its surface covered with mucus, containing epithelial or pus cells. When the deeper tissues are affected, a tumefaction of the parts may take place, from accumulation of the inflammatory products beneath the mucous membrane; which may be attended with great danger. Erosions or ulcers may occur, accompanied by ecchymosis of the membrane, and escape of blood into the secretions (due to rupture of the capillary vessels).

ETIOLOGY.

Predisposing Causes.

Badly nourished, cachectic subjects.—Constitutional vice.

Exciting Causes.

Chilling of the surface (by exposure).—Mechanical violence.—Inhalation of irritant gases, etc.—Exanthematous fevers (chiefly typhus). Blood poisons (diphtheria, syphilis, etc.).—Tonsillitis.—Erysipelas.—An accompanying influenza or bronchitis.

SYMPTOMS.

Rational.

The approach is insidious.
Sore throat and constriction exist.
Cough—is at first harsh and stridulous, becoming metallic as the disease increases.
Tenderness on pressure over the larynx.
Tickling sensation in the throat.
Dyspnœa.
Dysphagia.
Prolonged and wheezing inspiration and expiration, if the inflammation is severe. The patient is often unable to lie down on this account.
Expectoration—(if any) is tenacious at first, and, later on, is thick, purulent, and abundant.
Face—flushed at onset (the skin is usually hot and dry).
Countenance—pale and anxious as the disease increases.
Temperature—elevated—105°.
Pulse—hard and frequent.
Drowsiness, suffocation and cyanosis—from imperfect aëration of the blood.
Delirium—in severe cases.
Coma—in severe cases.
Death—in severe cases.

Physical.

Inspection—respiration at first is labored, but, later on, may become of a gurgling and gasping type.
Auscultation—a feeble vesicular murmur is heard over the lungs—which may be entirely absent.
On Laryngeal Examination.
The mucous membrane is found to be bright-red. If severe,

there may be œdema; the parts are red, swollen, and semi-transparent, and the œdema is usually on the ventricular folds. These changes may extend to the trachea, or remain in the mucous membrane of the larynx or of the epiglottis.

DIFFERENTIAL DIAGNOSIS.

Croupous laryngitis, diphtheria, œdema laryngis, spasmodic asthma, hysteria, laryngeal spasm, thoracic aneurism, may be mistaken for this disease. *In very young children*, it must be distinguished from croupous laryngitis, which is seen by the laryngoscope as a false membrane; but the history, constitutional symptoms, and physical examination will stamp either of the other diseases.

PROGNOSIS.

In adults, it depends upon the amount of œdema present.
In infancy, if it is severe, the disease is always fatal.

CAUSE OF DEATH.

Death may occur in a few hours from œdema glottidis, if the attack be severe; but if mild, five to eight days usually elapse before death occurs. It is caused from closure of the rima glottidis, from swelling of the mucous and submucous tissues, and it may also occur from pulmonary or cerebral congestion.

TREATMENT.

Venesection—only if the patient is robust, and there is a likelihood of syncope. A warm, moist, uniform temperature is essential. The temperature of the room should never be below 76° F. *Absolute rest to the larynx* is all-important. *Vapor inhalations* should be resorted to early in the disease and persevered with. *Quinine* should be given in large doses and cinchonism produced; for a child three years of age, gr. xx. may be administered during the first twenty-four hours, if it is suffering from a severe attack. If there is œdema, the parts should be scarified, or tracheotomy performed, if you are unable to scarify; but tracheotomy may cause the inflammation to extend to the trachea, and result in pneumonia and bronchitis.

CHRONIC CATARRHAL LARYNGITIS.

SYNONYM.

"Clergyman's sore throat."—Laryngeal Phthisis.

DEFINITION.

Is a chronic inflammation of the lining membrane of the larynx (which may be general, or partial, or manifest a tendency to remain stationary), and in which the vessels of the areolar tissue slightly participate.

MORBID ANATOMY.

When fully developed, the mucous surface is usually coated with mucus or pus, and is of a dark or grayish-red or blue color, due to ecchymosis, and may be softer or firmer than normal. The glands are enlarged and prominent; and there may also be erosions, fissures, ulcers, polypi, etc. Sub-mucous thickening and œdema may also occur, producing considerable narrowing of the glottis. The epiglottis may often be eroded.

ETIOLOGY.

Predisposing Causes.

Pharyngeal inflammations (from alcoholism or tobacco in excess).—Chronic bronchitis of old age.—It may be a primary disease, or a sequela of acute laryngitis.

Exciting Causes.

Irritating inhalations.—Morbid growths (laryngeal).—Pulmonary phthisis, syphilis, etc.—An extension of a laryngeal, catarrhal, or follicular faucitis (when the racemose glands of the larynx are principally affected).

SYMPTOMS.

Rational.

Voice.

Chronic hoarseness, or a husky whisper, or an ultimate loss of voice.

Cough.

Hoarse or stridulous.

Expectoration.

Muco-purulent or purulent (possibly streaked with blood).

Œdema—as shown by the laryngoscope.

Pain and tenderness on pressure over the larynx.

PHYSICAL.

Auscultation.

Moist râles over the larynx may be detected.

Laryngoscopic examination.

Shows general or partial hyperæmia of the lining membrane, and dilatation, or contraction of the larynx ; a muco-purulent secretion on the mucous membrane ; or, the membrane may be dry and shining in appearance.—The orifices of the glands may be seen as pale specks on the congested membrane, or as red circles. In syphilitic or phthisical patients, in addition to the above symptoms, there may be thickenings, ulcerations, or papillary excrescences.—Chronic œdema, of one or both of the aryteno-epiglottidean folds, is often observed in the chronic laryngitis of phthisis.

DIFFERENTIAL DIAGNOSIS.

From laryngeal growths; nervous affections; pulmonary phthisis (when examination of the lungs decides); and syphilis (where the history decides).

PROGNOSIS.

Depends upon the pathological associations.

Recovery is rare, if it occur in connection with pulmonary phthisis; the chances of recovery are better if it depend on syphilis, but the voice is permanently affected, as a rule.

Is usually good, unless complications exist. The disease is rarely fatal in itself.

TREATMENT.

Local internal applications should be frequently applied to the diseased tissues. A solution of nitrate of silver (gr. lx. to ʒ i. of water), or chloride of zinc (gr. xxx. to ʒ i. of glycerin), is often used with benefit.

Inhalations with either of the following prescriptions may be applied for five minutes at a time, three times a day : ℞. Ol. pini, ʒ ij.: Mag. carb., gr. ij.; Aquæ, ℥ ij.; Sig. one drachm in a pint of hot water (at 150° F.).—℞ Creasoti, ʒ iij. ; Glycerinæ, ʒ iij., Aquæ, ℥ iij ; or ℞ Ol. juniperis, ♏ l.; Mag. carb., gr. x.; Aquæ, ℥ ij.; or ℞ Acid. carbol., ʒ ss.; Glycerinæ, ʒ ij.; Aquæ, ℥ ij.

Nebulized liquids, or spray of either of the following drugs, should, like the inhalations, only be continued for five minutes at a time : alum, perchloride of iron, tannin, or sulphate of zinc, from gr. i.–xx. to the ounce of water.

Rest of voice is imperative, and the patient should be removed to a warm dry climate. If the disease is due to syphilis, *potassium iodide* should be administered in large doses, and the patient put under proper hygienic treatment ; or, if due to phthisis, the general treatment of phthisis is indicated.

ŒDEMA GLOTTIDIS.

DEFINITION.

Is a dropsical effusion, usually of inflammatory origin, in the areolar tissue above the vocal bands; or. in other words, an œdema of the upper portion of the larynx.

MORBID ANATOMY.

The changes that occur in this disease are principally observed during life; as, after death, owing to the disappearance of the greater part of the effusion, the mucous membrane is found to be wrinkled, and probably a very small quantity or only a trace of the effusion can be found.

On examination, during life, a tumor, about an inch or so in diameter, may be observed projecting, on one or both sides, into the cavity of the larynx and often into the pharynx, which may completely close the laryngeal opening. The mucous membrane may be either red or pale. The serous effusion commences in the loose cellular tissue beneath the mucous membrane, principally in the *aryteno-epiglottidean folds*, at the base of the epiglottis. When the tumor is pricked, a clear, turbid, or purulent fluid escapes.

Collapse of the tumor and wrinkling of the mucous membrane then take place.

ETIOLOGY.

The predisposing causes are: constitutional disturbance; any inflammation of adjacent tissue, or local irritation (as acute laryngitis); erysipelas; deep-seated cervical abscesses; acute tonsillitis; laryngeal ulceration; due to typhus, typhoid, scarlatina, and small-pox; Bright's disease; venous obstructions in cardiac diseases; and thoracic aneurism.

SYMPTOMS.

Rational.

Dyspnœa on inspiration, (often alarming).
No fever or constitutional disturbance.
Suffocative breathing (often the first indication).
Paroxysm of strangulation.
Cyanosis.
Veins prominent (due to obstructed venous return).
Drowsiness (due to approaching cyanosis).
Cold extremities.
Inclination to rid the upper part of the throat of some supposed secretion.

Physical.

The laryngoscope reveals two small tumors, about one-half an inch to one inch in length, which are at first smooth and round and possibly oval, situated at the ventricular folds, thus causing difficulty in laryngeal respiration.

DIFFERENTIAL DIAGNOSIS.

Is easy, on account of the circumstances under which it is developed, together with the sudden occurrence, and the peculiar character of the respiration; the absence of febrile symptoms, and the aid of the laryngoscope confirm the diagnosis.

PROGNOSIS

Is fatal, if not properly attended to.

TREATMENT.

Prompt surgical interference, as laryngotomy, tracheotomy, or scarification are the only means of relief. Medications are of no avail.

ACUTE CROUPOUS LARYNGITIS.

DEFINITION.

A fibrous inflammatory exudation of the larynx, which usually commences on the tonsils and often extends to the trachea and bronchi.

MORBID ANATOMY.

There exists on the mucous membrane a whitish or yellowish-white fibrinous layer, often covered with dots or lines. This membranous exudation may be in patches, or form a cylinder, extending into the trachea or bronchi. It may be firm in consistence, and adhere with great tenacity to the subjacent membrane, or it may be soft and easily separated. The surface is smooth, and adheres with great firmness to the vocal cords and upper part of the epiglottis (which possesses pavement epithelium). The membrane may be cast off in the form of a cylinder, or shreds, in consequence of the damming up of the secretion of the follicles, as well as by the serous exudation from the previously inflamed membranes. It may be expectorated, or undergo granular, fatty, or mucous degeneration, thus resembling a mucous fluid; therefore, in the latter case, no membranous exudation is observed. Under the microscope it will be seen that there are generally pus and rarely epithelial cells. It is questionable whether these are changed epithelial cells or white globules. The mucous membrane, like all inflamed tissues, contains a varying amount of these cells. As the exudation is cast off, the surface epithelium is quickly repaired, and the laryngeal membrane returns to its normal condition.

STAGES AND DANGERS OF THE DISEASE.

1ST STAGE—Precursory or catarrhal. There is danger of the formation of a membrane in the trachea and larynx, if the inflammation continue downward.

2D STAGE—Development. There is danger of inflammation continuing to the minute bronchi.

3D STAGE—Suffocation or collapse. There is danger to the patient from exhaustion and carbonic acid poisoning.

ETIOLOGY.

Occurs between dentition and puberty. Rarely in very young infants or adults.

PREDISPOSING CAUSES.

Occurs in delicate, weakly, ill-fed children.

EXCITING CAUSES.

From exposure to cold and moisture.—From sudden alteration in temperature.—From irritation of hot water, etc., applied to the larynx.—Follows eczematous eruptions on the head and face, and frequently measles, scarlatina, and small-pox.

SYMPTOMS.

PRECURSORY OR CATARRHAL STAGE.

Rational.

Catarrh—mucous membrane red and tumefied.
Slight hoarseness.
Cough—noisy, high-pitched, stridulous.
Crowing noise after the cough.
Dysphagia—(puts the hand to the throat).
Pulse—accelerated, full, and hard.
Temperature of axilla—102° to 105°.
Fever—intermittent.
Redness of the face.
Dyspnœa—most severe at night.

Physical.

Inspection—difficult respiration.

STAGE OF DEVELOPMENT.

Rational.

Aphonia—speaks or cries in a whisper.
Cough—suppressed and more stridulous.
No expectoration.
Head thrown back (an evidence of dyspnœa).

Physical.

Inspection—difficult respiration; during inspiration there is a contraction of the lower part of the chest and sinking above the clavicles. Epigastrium is depressed, and there is arrested expansion of ribs.

Auscultation—feeble vesicular murmur.

STAGE OF SUFFOCATION OR COLLAPSE.

Rational.

Nostrils—dilated.
Great restlessness.
Countenance—pale and livid (expression of fear often present).
Pulse—rapid and feeble.
Temperature—falls below normal.
Cold extremities.
Drowsiness.
Gasping.
Cyanosis.
Paroxysmal dyspnœa.
Coma
Death.

DIFFERENTIAL DIAGNOSIS.

From simple catarrh of the larynx; (spasmodic or pseudo croup) in nervous subjects; purely spasmodic affections of the larynx (in each of which the spasm gives rise to croupy symptoms); diphtheria involving the larynx.

PROGNOSIS.

In childhood, this is the most fatal of all the diseases (especially if membranous exudation occurs on the tonsils and epiglottis).

Favorable signs—if the paroxysms of dyspnœa are diminished in frequency and severity; if the breathing is not so distressed; if there is a gradual return of voice, and a moist sound with the cough.

A fatal result is indicated if the dyspnœa become more frequent and violent; the cough less powerful and stridulous; and when there is marked cyanosis and drowsiness.

The recovery is slow. It may be weeks before the patient regains

the use of the voice, and violent dyspnœa may occur during the whole period of recovery.

TREATMENT.

In the very early stage give *turpeth-mineral, Hyd. sulph. flavæ, gr. ij.*, or (as it is better known) "German croup powder." Patient should be placed in a large, *well-ventilated room*, with a temperature about 75 to 80°; and the air should be moistened by steam, etc., as in other laryngeal affections. Administer *Quininc, gr. xxx.*, in gr. v. doses to a child to arrest inflammation. A moist, nutritious diet should be given from the onset of the disease, together with *stimulants* if a failure of the vital power become apparent.

Tracheotomy (?) is only a temporary relief, and must be done early in the disease or not at all.

BRONCHITIS.

DEFINITION.

Is an inflammation of the mucous membrane of the trachea and bronchi, which may vary in extent, intensity and duration.

VARIETIES.

ACUTE CATARRHAL.—This form may be simple or capillary; and localized or general. It occurs in infancy and old age, from acute catarrhal inflammation, typhus, and measles; first affecting the large tubes, and by passing downward to the smallest tubes, often becomes capillary in form. In middle life, it affects the larger bronchi.

CHRONIC CATARRHAL.—This is a secondary affection. Is a low grade of inflammation of the bronchial mucous membrane, and a disease of adult life, having a tendency to recur, with increased vigor, till it becomes permanent.

CROUPOUS OR PLASTIC.—This may be acute or chronic. This is a very rare form of the disease. Is met with in the young adult, principally in the female, and those of feeble or delicate constitutions. In acute fatal cases, it only lasts from three to ten days; but, in

those likely to recover, its duration is about fourteen days. Complete recovery is, however. exceedingly rare.

ACUTE CATARRHAL BRONCHITIS.

MORBID ANATOMY.

This disease is the continuation of an inflammation which usually commences in the nasal, pharyngeal, or laryngeal mucous membrane; or the inflammation may commence in the alveoli and extend to the smaller tubes. The mucous membrane is found to be swollen and red, softer and more moist than normal, and occasionally ecchymotic. A clear transparent mucus is at first contained in the bronchi, which, as the disease progresses, becomes opaque, whitish, yellowish, greenish, or muco-purulent; this change is owing to an increased number of cells contained in the fluid (which are pus cells), and also to the presence of epithelium (often of the ciliated variety).

On a post-mortem examination, the presence of mucus or muco-pus in the tubes may be the only evidence of bronchitis; these same changes may exist whether the large or small tubes are affected. Yellow spots may be found on the surface of the lung, occurring in young, feeble children, from gravitation of mucus or muco-pus from the larger into the smaller tubes. The accumulation of this mucus or muco-pus may produce a temporary obstruction, and distention of the alveoli, which may be mistaken for vesicular emphysema.

ETIOLOGY.

Predisposing Causes.

Infancy and old age.—Indulgence in enervating habits.—Debility, from any cause.—Constitutional disease (as gout, syphilis, etc.).—Chronic pulmonary affections (phthisis, emphysema, etc.).—Bad air.

Exciting Causes.

Chilling of the surface of the body.—Secondary result of blood poisoning. — Irritation, produced by substances inhaled.—Atmospheric conditions.—Heart lesions (as mitral stenosis or

insufficiency, or tricuspid regurgitation). — Pressure on the aorta below the bronchial arteries, or from fluid in the peritoneal cavity, or gas in the intestines pressing the blood back in the abdominal aorta, thus causing congestion of the lungs and the bronchial tubes.

SYMPTOMS.

Rational.

Stage of Invasion.

Coryza. — Uneasy sensation in the frontal sinuses, extending down the nasal passage to the larynx and trachea.—Aching in the back and limbs; convulsions may occur in children.

Pain and discomfort behind the sternum.

Sense of soreness on coughing.

Sense of constriction in the chest.

Cough.

In the early stages is dry or hacking.—In the later is loose in character.

Expectoration.

Frothy mucous, muco-purulent, or purulent.

Physical.

Inspection and *palpation* are negative.

Auscultation—Sibilant and sonorous râles (in the dry stages); or large and small mucous râles after the disease is fully developed (which change their position on coughing).

DIFFERENTIAL DIAGNOSIS.

From pneumonia and pleurisy; but the symptoms of the stage of invasion and the physical signs are sufficient to distinguish it from either.

PROGNOSIS.

Never destroys life directly, unless it occur in very young children; as it usually terminates in resolution, or becomes chronic The inflammation may, however, sometimes extend to the smaller tubes, resulting in capillary bronchitis or lobar pneumonia.

TREATMENT.

In the stage of invasion, a Dover's powder, and a warm bath at

night, followed in the morning by a brisk saline cathartic (for children a full dose of castor oil), may be all that will be required to avert the attack. The patient must be kept in a warm room of equal temperature for a few days.

In the early stage, great benefit is derived from *inhalations* of the vapor of molasses and water; or from mustard poultices applied to the upper part of the chest.

Quinine gr. viij–x. should be given daily if the disease is likely to pass into the chronic stage, or to extend to the smaller bronchi. *Oleum morrhuæ, with lime-water*, should also be given; but if it occur with gout or rheumatism, give *colchicum with alkalies.*

ACUTE CAPILLARY BRONCHITIS.

DEFINITION.

It is an extension of acute catarrhal inflammation into the small-sized bronchial tubes, and may be localized or diffused.

Localized—if the inflammation is limited to the smaller tubes.

Diffused—when the inflammation attacks the lining membrane of all the bronchial tubes.

MORBID ANATOMY.

Is the same as in acute catarrhal bronchitis.

ETIOLOGY.

The same as in acute catarrhal bronchitis.

SYMPTOMS.

Rational.

Dyspnœa—marked (patient unable to lie down).
Recurring chills.
Febrile symptoms often well marked. Temperature 100° to 103°.
Pulse 100 to 130, and feeble.
Respiration—labored and frequent; 60 to 70 per minute.
Cough—violent at first; is usually hacking in character.
Expectoration—at first, of a thick tenacious mucus; later on, it becomes more abundant, less tenacious, thin, and frothy
Exhaustion—marked.

Countenance—anxious and flushed; in later stages, *livid.*
Great restlessness—(later on in the disease).
Pulse—becomes small and thready as death approaches; is always raised in frequency.
Body—is covered with a cold, clammy sweat as death approaches.
Delirium—muttering.
Coma—partial.
Death—from apnœa (from imperfect oxygenation of the blood).

PHYSICAL.

Percussion—is exaggerated in the infra-clavicular regions (unless œdema exist).
Auscultation—diminished respiratory murmur. — High-pitched *sibilant* râles.—*Subcrepitant* râles, as the disease advances.

DIFFERENTIAL DIAGNOSIS.

From pneumonia; pulmonary œdema; acute and chronic phthisis.

PROGNOSIS.

When general capillary bronchitis occurs in infancy or old age, or follows phthisis, Bright's, heart disease, or any acute blood disease, it is always attended with great danger.

If there is difficulty of expectoration, signs of accumulation in the bronchial tubes, shallow breathing, cessation of cough, dyspnœa, apnœa, or failure of heart's power, the prognosis is unfavorable.

TREATMENT.

The patient must be kept in bed the surface of the body covered with flannel, the temperature of the room be about 70°, and the air moistened with steam. The diet should be of a concentrated nutritious nature as milk, eggs, etc. *Dry cups* must be applied to the chest which should afterwards be covered with an oil-skin jacket. *Steam inhalations* should be used freely, as it increases the secretion and facilitates expectoration.

Give *muriate of ammonia and chlorate of potassium* gr. v.–x., every two hours to an adult (gr. ij. to a child two years of age).

If expectoration is entirely absent, give stimulating emetics.

Should the pulse become small and thready, give *quinine and brandy. Stimulants* require to be administered judiciously early in the disease and continued.

If there is spasm of the bronchial tubes, full doses of *hydrocyanic acid* may markedly relieve, *but never give opium.*

CHRONIC CATARRHAL BRONCHITIS.

DEFINITION.

Is a low grade of inflammation of the bronchial mucous membrane, having a tendency to recur with increased severity and duration, till it becomes permanent.

It may be primary or secondary. *Primary*, when it results from external causes; and *secondary*, when it results from some previously existing or constitutional dyscrasia.

MORBID ANATOMY.

The inflammation may be limited to the large bronchi, or it may extend into the capillary tubes. The bronchial mucous membrane has a gray or reddish-blue color, and, in chronic cases, is often hypertrophied. Its glands are enlarged and prominent, and their ducts so increased in size that their mouths are readily visible. In the early stage, the other coats of the bronchial tubes may be weak or yielding; and later on, there may be thickening and induration. The results of chronic bronchitis are *dilatation* and *stenosis* of the bronchial tubes; an accumulation of secretion in a state of cheesy degeneration (obstructing their calibre); and pulmonary emphysema and induration of the adjacent lung-tissue.

ETIOLOGY.

Predisposing Causes.

Exposure to cold or wet.
Bad air.
Constitutional diseases (gout, etc.).

Exciting Causes.

Irritation by substances inhaled.
Mitral stenosis.
Chronic alcoholismus.

SYMPTOMS.

Rational.

In Early Stage, or simplest form.

Cough—at first, may be slight.

Expectoration—muco-purulent, which comes on in the winter and disappears in the summer, and ultimately becomes permanent.

Later on in the Disease.

Cough—is violent, (especially in the morning).

Expectoration — tenacious and scanty, or thin, semi-transparent, and abundant, sometimes streaked with blood and difficult to expectorate; varies from an ashy yellow to a dark-green color, and sinks in water.

Emaciation—(may occur).

Fever—(may occur).

Night sweats—(may occur).

Dyspnœa.

Pulse—is normal.

Temperature—is normal (except where there is a fœtid breath).

Soreness behind the sternum (increased by coughing).

Physical.

Inspection—respiration is accelerated and labored.

Palpation—vocal fremitus may be normal, exaggerated, diminished, or absent.

Percussion—normal, or temporary dullness.

Auscultation—vesicular murmur is more or less deficient, and the respiratory sound is coarse, loud, and harsh, with prolonged expiration. There may be large or small mucous râles which are altered in character and position by coughing, and by a full inspiration. Vocal resonance is normal, diminished, or slightly exaggerated.

DIFFERENTIAL DIAGNOSIS.

From pleuritic effusions; pneumonic consolidation; and pulmonary phthisis.

PROGNOSIS.

This disease rarely destroys life directly; but, if associated with any pulmonary affection, the condition of the patient is serious, on account of the liability to obstruction from the accumulation of the secretion in the bronchial tubes. When it occurs in persons past middle life, recovery is rare.

TREATMENT.

Remove the patient from every source of bronchial irritation, and guard against exposure to changes of temperature. Flannel should be worn next to the chest. Keep the patient in the house in bad weather. See that the apartments are well ventilated and at a temperature of about 65° to 70° F. The patient must not be allowed to expose himself to the night-air or cold winds, and, if possible, should be sent to a warm, dry climate. If there is severe emaciation, a long sea voyage is advisable. The diet should always be nutritious, and a *moderate amount of stimulants* will be necessary.

If a rheumatic or gouty diathesis exist, then give *colchicum and alkalies*. *Iodide of potassium* should be administered internally if the disease be associated with pulmonary emphysema.

If there be anæmia, *iron* is indicated and a general tonic treatment, when quinine, mineral acids, bitter vegetable infusions may be combined with iron. If the bronchial secretions are excessive, *steam inhalations* of tar, creasote, and naphtha are of great service. When the expectoration is deficient, *stimulating expectorants*, such as *senega*, *serpentaria*, *camphor*, *tincture of benzoin*, combined with *carbonate of potash*, *soda*, etc., are indicated.

If the bronchial mucous membrane is irritable, the secretion scanty, and the cough paroxysmal, *opium*, *hydrocyanic acid*, *hyoscyamus*, *belladonna*, or *conium* should be administered in full doses.

If the bronchial secretion accumulate in the larger bronchi and cannot be expectorated, emetics may be employed.

CROUPOUS BRONCHITIS.

SYNONYM.

"Plastic bronchitis."

MORBID ANATOMY.

It differs from catarrhal bronchitis, since plastic material is poured out into the tubes in the form of casts, which are either solid or hollow, according as the large or small tubes are affected, and are of a whitish color, sometimes dotted over with blood spots.

In the *chronic form*, the membranous exudation occurs only over a circumscribed portion of the bronchial membrane; in the *acute*, it is distributed over a greater portion of the bronchi. The membrane may be firmly adherent, or loosely attached to the mucous surface. The casts under the *microscope* consist of fibrillated fibrin, abundant granular matter, exudation corpuscles, and ovoid cells.

ETIOLOGY.

It is most frequently met with in the young adult, especially in females, and in those of a feeble and delicate constitution. It is said to be due to some diathetic state.

It may be acute or chronic, but both forms are rare; the acute is the most infrequent.

SYMPTOMS.

Rational.

Fever.

Cough—which is hoarse and ringing in character.

Dyspnœa—(often severe).

Hæmoptysis.

Expectoration—consists of *casts of the bronchial tubes* (often resembles trees with short branches).

Death—may result from asphyxia.

Physical.

Percussion—may be normal, extra resonant, or dull.

Auscultation—Respiratory murmur is feeble ; or absent, if the bronchial tubes are obstructed. Dry or moist râles are usually present.

DIFFERENTIAL DIAGNOSIS.

From acute catarrhal bronchitis ; pneumonia ; or pleurisy.

PROGNOSIS.

In the acute form (which lasts from three to ten days) more than half die. In the chronic form, which lasts from ten to fourteen days, if it is not complicated, the prognosis is good as regards life, but having once occurred is apt to return. Complete recovery is rare. It is very likely to lead to pneumonia and phthisis.

TREATMENT.

In the acute form, the treatment is the same as in croupous laryngitis. In the chronic form, alkaline *steam inhalations* should be used during the paroxysm, and the patient kept in a warm equable temperature. During the interval, *iodide of potassium, quinine, iron, and cod-liver oil* have been recommended. If the paroxysms return, the patient should be sent to a warm climate, or on a long sea voyage.

ASTHMA.

Definition.

Is a condition produced by spasm of the involuntary muscular fibre of the bronchial tubes.

MORBID ANATOMY.

This disease is looked upon as a spasmodic affection of the bronchi causing dyspnœa. These bronchial contractions are due to a reflex nervous action ; and the disease may be regarded as a neurosis, dependent upon the existence of a peculiar diathesis. Catarrhal symptoms may be wanting, and, when present, may precede the paroxysm. These attacks may occur in those suffering from organic diseases of the heart, or lungs, but true asthma is spasmodic.

ETIOLOGY.

Hereditary predisposition ; irritation of the mucous membrane of the bronchi by certain atmospheric changes ; reflex irritation, from abnormal conditions of the nervous system, or of organs ; or as a complication of some other disease.

SYMPTOMS.

Rational.

Premonitory symptoms (peculiar to the individual) are present in some cases, as drowsiness, wakefulness, headache; etc.
Dyspnœa—intense and of sudden onset.
Respiration—wheezing.
Posture—sitting (*arms braced, mouth open*).
Face—red, or livid, and anxious.
Extremities—blue and shrunken in prolonged cases.
Cough and expectoration (after the attack) which may consist of mucous or be profuse and watery.
Sense of extreme exhaustion—after the attack has subsided.

Physical.

Percussion—exaggerated.
Auscultation—high-pitched, sibilant, and sonorous râles.
Respiratory murmur—is jerking or exaggerated and usually short.

DIFFERENTIAL DIAGNOSIS.

From acute capillary bronchitis ; pulmonary œdema ; pulmonary congestion ; hydro-thorax ; angina pectoris ; and laryngeal obstruction.

PROGNOSIS.

Is seldom, if ever, directly a cause of death. This class of patients are generally long-lived, as they have to attend most rigidly to all the principles of hygiene.

TREATMENT.

It should be borne in mind that you must first relieve the paroxysms, and then prevent their occurrence. In order to accomplish this end, it will be your duty first to ascertain the cause,

and then, by overcoming the cause, you may relieve the paroxysm at once. If you are unable to remove the cause, or the paroxysm is not relieved, place the patient in the easiest position possible. The best remedies to use in this disease are *stramonium*, in the form of cigarettes; *chloroform; hyoscyamus; ergot; ether; cannabis indica, opium* or *atropine;* and the fumes of burning nitre paper.

Loomis advises *opium, in full doses* ($\frac{1}{2}$ gr. morph. sulph. hypodermically); but very good results have been obtained with the prescriptions which will be found at the end of this volume, and should be tried, especially *hyoscyamus and ergot* for the first two or three days; if they have not the effect of relieving it for some considerable time, the other remedies might be resorted to. Should there be anæmia, or the patient be poorly nourished, *ol. morrhuæ and iron* should be administered at intervals. *Quinine, gr. v.-x. daily*, often prevents the paroxysms, and it must be continued regularly, or else the paroxysms will return.

WHOOPING COUGH.

SYNONYM.

Pertussis.

DEFINITION.

It is an infectious and portable disease, dependent upon a specific poison; characterized by a paroxysmal cough, consisting of a series of short, spasmodic, forcible expirations, followed by a deep, prolonged inspiration, attended with a peculiar sonorous sound called the "whoop" or "kink," the paroxysms terminating in expectoration or vomiting.

MORBID ANATOMY.

The principal changes are those of catarrhal bronchitis. It is considered by many prominent physicians to be of nervous origin; and that either an inflammation of the pneumogastric nerve or congestion of the medulla oblongata exists. The complications are lobular collapse, lobular emphysema, bronchial dilatation, and catarrhal pneumonia.

STAGES.

It has three stages, catarrhal, spasmodic, and decline.

SYMPTOMS.

Rational.

In the Catarrhal Stage.

Marked naso-pharyngeal and bronchial catarrh.
Coryza.
Cough—paroxysmal in character.
Expectoration—abundant, (tenacious, viscid, transparent mucus).

In the Spasmodic Stage.

Cough—severe and distressing, there is at first a long, clear, piping, inspiratory sound ; followed by convulsive and forcible expiratory puffs and succeeded by a whoop.
Face—becomes red or purplish during the paroxysm.
Eyes—protrude during paroxysm.
Vomiting may follow paroxysm.
Bleeding from nose, mouth, and ear may occur.

In the Stage of Decline.

Paroxysms gradually diminish in frequency and severity.
Expectoration is less difficult (becomes more purulent, and, after a while, ceases altogether).

Physical.

Auscultation—during the paroxysm, the respiratory murmur is feeble or absent. Sibilant and sonorous râles (which during the interval are mucous in character).

DIFFERENTIAL DIAGNOSIS.

In the early stages, its diagnosis is uncertain ; but if there is a violent spasmodic cough, the fever excessive and prolonged, and the disease is prevalent, its existence may be suspected. Its peculiar cough distinguishes it from all other diseases.

PROGNOSIS.

Is always a serious disease, although rarely directly fatal ; but

it frequently causes death indirectly. Fatal results are due to complications.

TREATMENT.

In order to relieve the paroxysms of coughing, the most effective remedies are *belladonna, hyoscyamus, cannabis indica, chloroform*, and *musk*, which should be administered in small doses. The patient should guard against undue exposure; his clothing should be warm. During fine, warm weather, the patient should be in the open air as much as possible; but damp, cold weather, and exposure to draughts must be strictly avoided. The diet should be simple, and the bowels carefully attended to. In obstinate cases and in convalescence, a *change of air*, if only for a short distance, often proves very beneficial. If possible, mountain, or sea air, or pure country air, should be chosen, as it acts favorably by removing irritation of the nervous system, and completing restoration to health.

EMPHYSEMA.

DEFINITION.

Is an abnormal accumulation of air within the air-cells; or an infiltration of air into the sub-pleural cavity, or interstitial connective-tissue, due to rupture of the air vesicles.

VARIETIES.

Vesicular—where the *air-cells alone* are over-distended and *contain an excess of air.*

Interlobular or *interstitial*—where the *air* has escaped *between the lobules* of the lung (through rupture of the air-cells).

MORBID ANATOMY.

Before opening the body you will notice the "*barrel-shaped*" chest, emaciation, and the emphysematous countenance.

On opening the chest, the lungs fail to collapse, and, in bad cases, protrude from the chest; they frequently bear the impression of the ribs, and have blebs on the surface (due to an interstitial emphysema).

The specific gravity of the lung is greatly diminished; they are paler in color than normal, and crepitate less than normal. The bronchi are partially filled with sticky mucus, and spaces may be seen, by the eye (when a section is made), in the lung substance, due to breaking down of the air-cells, thus allowing of a communication with each other. The heart will be found displaced, and the *right heart* is usually hypertrophied. Tricuspid regurgitation is not infrequent. There is often found the "*nutmeg*" liver, and the other organs may be in a state of fatty degeneration.

When one of the lungs is removed, inflated, injected through the pulmonary and bronchial vessels, and dried, *a section* will show (1st) abnormal spaces due to rupture of the air vesicles; (2d) obliteration of the blood-vessels in the septa between the air-cells; (3d) destruction of the septa between the air-cells, due to fatty degeneration, from poor nutrition, on account of destruction of the blood-vessels.

ETIOLOGY.

Primary Causes.

Forced inspiratory and expiratory efforts.—Severe coughing or straining.—Hereditary, or acquired impairment of the elasticity of the lung tissue.—Abnormal growth of the chest-walls.

Secondary or Compensatory Causes.

Emphysema of the immediate lung tissue, around small portions of the lung, rendered inexpansible from disease.

Vicarious Emphysema.

Emphysema, of the healthy lung, may be produced where large portions of the lung tissue are consolidated, or impaired by disease, as in atelectasis, from obstruction of a small bronchus, a lobar pneumonia, or pulmonary infarction.

Interlobular emphysema may follow the vesicular form (by rupture), or perforation of the lung tissue (from traumatism, foreign bodies, etc.).

SYMPTOMS.

Rational.

Dyspnœa.

Increased by exercise.—Increased by attacks of bronchitis.—Increased by attacks of asthma.—Increased by cold climate.

Expectoration.

Of a pearly, tenacious mucous (due to an accompanying bronchitis).

Countenance.

Dusky—Puffy, even where the body is emaciated.—The nostrils are distended, and vascular, and expand with inspiration. —Angles of mouth are drawn down.—Jugular veins are distended.

General Condition.

Feeble voice.—Emaciation.—Stooping attitude in walking.—Gradual loss of strength.—Low temperature.—Feeble pulse.—Vertigo, after coughing.—Œdema of feet and ankles (in late stage) due to tricuspid regurgitation.—Exhausting cough.

PHYSICAL.

Inspection Reveals

Prominence of the sternum.—Widening of the intercostal spaces.—*Round or "barrel shaped" chest.*—No actual expansion, but a *rising* and *falling movement* of the sternum during respiration.—Displacement of the apex-beat.—Abdominal breathing.—Muscles of the neck very prominent.

Palpation.

Variable—vocal fremitus is usually diminished, except in the senile form, when it is increased.

Percussion.

Vesiculo-tympanitic (due to too much air).

Asucultation.

Inspiratory sound, is feeble, or suppressed.—*Prolonged, low-pitched, expiratory sound* (due to loss of elasticity of the lung-tissue).—*Crackling sound* like the crumpling of parchment.

DIFFERENTIAL DIAGNOSIS.

From pneumo-thorax only.

COMPLICATIONS.

Heart disease, (chiefly of the right heart); bronchitis (acute attacks).

Fatty degeneration of the organs throughout the body, and dis-

ease of the kidney, or liver (due to the interference of the general circulation).

PROGNOSIS.

It is an incurable disease; is modified by the extent of the disease, and by its complications; the most frequent being bronchitis, which is always an extremely grave affection, when the smaller tubes are attacked.

TREATMENT.

Iron should be given daily with the meals for a long period, in the form of the *ethereal tincture of the acetate;* and, also *sulphate of quinine* in small doses with the iron. If there should be dyspeptic symptoms, bitter vegetable infusions should be given; and *oleum morrhuæ* for the emaciation that is present. *Stimulants* are often of great benefit when taken in small quantities during, or shortly after meals. The diet should be most nutritious; principally animal food. The stomach should not be overladen. The food should not be bulky or watery in character, but as digestible as possible. Small quantity of liquids should be used at a time. The patient should live in the open air, but not expose himself to cold; and all violent exercise should be avoided. The complications should be treated as they arise; but the most persistent is bronchitis, and, when this occurs, *iodide of potassium* should be administered, in gr. v.–xx., three times a day, and continued for a long period. The other complications will be treated under their respective headings.

PULMONARY ŒDEMA.

DEFINITION.

Is a serous transudation from the pulmonary blood-vessels, dependent either upon some local cause, or an altered state of the blood.

MORBID ANATOMY.

Serum will be found in the cavity of the alveoli, and the interstitial-tissue of the lungs. If the œdema be associated with congestion, the serum will be blood-stained; otherwise it will be of a

clear color. On opening the thorax, the lungs do not collapse. That part of the lung where the œdema is present will be paler than normal, unless congestion be present. In the œdematous portion of the lung, if it pit on pressure, it proves that the elasticity of the lung has been diminished. *On section*, serum exudes; which is usually frothy, unless the air-cells are filled with serum. Œdema may occur in any portion of the lung, but it is usually in the most dependent part.

In order to determine whether the œdema has occurred before or after death, it will be necessary to know what the physical signs and symptoms were before death took place.

ETIOLOGY.

General hydræmia (as in purpura, Bright's disease, scurvy, etc.).

Pulmonary congestion (dependent upon obstructed return circulation through the heart).

Enfeebled heart's action (chiefly in the aged).

Long confinement to one position (as in fevers, debility, etc.), because the blood always goes to the most dependent part of the body.

Inflammatory conditions of the lung tissue (as in pneumonia, tuberculosis, capillary bronchitis), due to passive hyperæmia or stasis.

SYMPTOMS.

Rational.

Dyspnœa—is often sudden and severe.

Respiration—is increased in frequency.

Cough.

Expectoration—watery, and slightly blood-stained (if pulmonary congestion exist).

Temperature—normal.

Pulse—if increased in frequency, is feeble.

Cyanotic Condition (*if œdema is excessive*) *as shown by*

Blueness of the face.—Blueness of the extremities.—Coldness of the limbs.—Delirium.—Drowsiness.—Coma.

Physical.

Percussion—there is dullness over the seat of the œdema which is

usually at the most dependent portion of the lung. This dullness never reaches the condition of flatness, nor changes position.

Auscultation—bubbling râles over the seat of the œdema, heard with inspiration and the commencement of expiration. These râles have a liquid character, and are thus to be distinguished from the crepitant râle. Respiratory murmur is feeble, or absent, or harsh.

DIFFERENTIAL DIAGNOSIS.

From pneumonia (1st stage) — hydro-thorax — capillary bronchitis—and pulmonary congestion.

PROGNOSIS.

Is bad, if it depend upon the general dropsy of renal or cardiac diseases.

Is bad, if complicating an extensive pneumonia.

Is bad, if extreme cyanotic symptoms appear.

Is bad, if dependent on exhaustive diseases.

TREATMENT.

If it occur in connection with Bright's disease, increase the excretory functions of the kidneys, bowels, and skin with hydragogue cathartics, diuretics, and diaphoretics, and crowd them to the fullest extent.

Apply dry cups to the thorax. *Stimulants* should be given, if it occur in connection with typhoid and typus fever. Any means which will relieve or arrest the congestion are of benefit.

If the œdema be due to the patient remaining too long in one position, then move him constantly to prevent the gravitation of the fluid to the same dependent part; and endeavor to have the lungs emptied as frequently and fully as possible.

PULMONARY CONGESTION.

DEFINITION.

Is an excess of blood in the circulation of the lung.

Varieties.

Active Hyperœmia.

Active—associated with dilated capillaries, increased flow of blood to the part, and an increased rapidity of the current.

Passive Hyperœmia.

Passive, or "congestion"—associated with dilated capillaries, increased flow of blood to the part, and a slow current.

Hypostatic congestion—is that form of pulmonary congestion which is due to gravitation, and associated with intra-cellular œdema.

Compensatory or collateral congestion—is that form of pulmonary congestion which is due to obstruction in some other part of the lung.

Splenization of the lung—is that form of pulmonary congestion, which may be superficial or central; is associated with interstitial œdema, with red or yellow spots throughout the lung; and which resembles the spleen in appearance.

Brown induration of the lung—is that form of pulmonary congestion which is characterized by pigmentation; as a result of chronic passive hyperæmia, from mitral obstruction or regurgitation.

MORBID ANATOMY.

When the condition constitutes hyperæmia, the lungs are distended, of a dark-red color, slightly crepitating, and heavier and less elastic than normal. *On section*, dark blood flows freely from the cut surface; but the lung-tissue is still of a dark color, due to blood remaining in the capillary vessels.

Pulmonary hyperœmia may be active or passive, it is simply an increased quantity of blood in the capillaries, and may be due to increased heart's action, or obstruction to the return of blood to the heart, or local interference with the pulmonary circulation. The various conditions which the lung may assume are as follows:

Splenization.—In this condition the lung is of a dark reddish-blue, brown, or black color, airless, firmer than normal, and crepitates imperfectly. *On section*, a dark fluid oozes from its surface, but not so freely as in hyperæmia.

Hypostatic congestion.—This occurs in persons suffering from diseases which confine them to their bed for a long time. This form closely resembles splenization, but has neither the doughy feel, nor the whitish or reddish points.

Compensatory Congestion.—This is due to obstructed pulmonary circulation, (as in pneumonia) and occurs also in collapsed lung-tissue.

Brown induration.—This is epecially connected with *mitral obstruction*, or *regurgitation.* The lung will be found to be distended, firm, heavy, slightly moist, and dotted over with yellowish or brownish spots; these spots are due to old *blood extravasations* which have undergone granular pigmentary degeneration. The capillaries are enlarged. Under the *microscope*, the air-cells are found to contain large cells which have undergone more or less pigmentation.

ETIOLOGY.

Causes of Active Hyperœmia.

Violent heart's action.—Accelerated heart's action.—Excessive exercise, especially in those whose chest is small.—Inhalation of too stimulating air.—Too rarefied air.

Causes of Passive Hyperœmia.

Feeble heart's action (due to obstruction to the return circulation of the lungs).—Mitral disease.—Pressure on the pulmonary veins.—Fluid in the pleura, aneurism, cancer, tumors, collapsed lung (by impeding the venous return from the lungs).

Hypostatic Congestion.

Is dependent on gravitation, and occurs in those who have been long confined to one position.—Is associated with *intracellular œdema* of the lung.

Compensatory Congestion.

Is the result of obstruction in the circulation in some other part of the lung. Occurs in connection with pneumonia, mediastinal tumors, and collapsed lung.

Splenization.

Lung resembles spleen-tissue.—Is due to hyperæmia, with *interstitial œdema* superadded.—Occurs chiefly in connection with typhoid and typhus fever. Is of slow development.

Brown Induration.

Is a *pigmented condition* of the lung, due to obstruction or regurgitation at the mitral valve.

SYMPTOMS.

RATIONAL.

Dyspnœa, (very marked if pulmonary œdema exist).
Cough.
Expectoration (blood-stained and watery).
Symptoms of impaired function of lung-tissue.

PHYSICAL.

Palpation.—Increased vocal fremitus.
Percussion.—Dull over the seat of the disease (on account of œdema).
Auscultation.—Respiration feeble, or harsh; liquid râles, if œdema exist.

DIFFERENTIAL DIAGNOSIS.

From pulmonary œdema; spasmodic asthma; first stage of pneumonia; and capillary bronchitis.

PROGNOSIS.

In active hyperœmia—of a rapid type; terminates either in recovery, pneumonia, or pulmonary hemorrhage.
In passive hyperœmia—depends upon the complications; especially heart-diseases.
In brown induration—the prognosis is uncertain.
In splenization—the results are unfavorable.

TREATMENT.

When the congestion is active and comes on suddenly, the quantity of blood in the vessels must be lessened. *Wet* or *dry cups* should then be applied over the entire chest, or, by opening of the vein in the arm, the general system may be depleted. In passive congestion, the pulmonary circulation must be regulated. If the heart's action be weak, *stimulants* must be resorted

to, but should the heart's action be too forcible, give *aconite* in full doses. If it be associated with pulmonary œdema, administer *cathartics.*

PULMONARY APOPLEXY.

VARIETIES.

Circumscribed, or "pulmonary infarction," when the lung tissue is not lacerated.

Diffused, or "pulmonary apoplexy," when the parenchyma of the lung is lacerated.

MORBID ANATOMY.

In the circumscribed variety, the lungs contain hemorrhagic nodular infarctions, which may be of a dark-red, or black color, containing no air. These infarctions may be hard or soft, according to their age, and are recognized by their firmness when pressure is made. They may be from one to four inches only, but are usually about one inch in diameter. They are found most frequently in the posterior portion of the lower lobe of the lung. These nodules are, as a rule, formed by the escape of blood from the capillaries of the air-cells into the cavity of the alveoli, and minute bronchi, some of which is retained in these cavities as the blood coagulates; the capillaries around the alveoli are found to be filled with blood, but there is no rupture of the lung-tissue. Resolution is the most frequent change that occurs; the infarction becoming either absorbed or expectorated.

As a second result, these infarctions may remain as dark pigmented, cicatricial spots, impermeable to air; they may excite inflammation of a pneumonic character, which may terminate in gangrene; or the nodules may become gangrenous.

If the infarctions occur in connection with pyæmia, they may terminate in gangrene, or in the formation of abscess.

If the infarctions be near the surface, the pleura may be lacerated. In case the diffuse variety exists, the cavity made in the lung tissue by the extravasation is of considerable size, and the coagulated blood resembles a blood-clot. These extravasations are diffused and are much larger than the circumscribed variety.

They may prove fatal at once, if the pleura be perforated. If the patient survive the shock, recovery usually takes place; either by adhesion of the torn surfaces, or a connective-tissue capsule being formed around the clot, which undergoes a cheesy, chalky, or pigmented degeneration, and remains imbedded in the lung tissue.

ETIOLOGY.

Causes of the Circumscribed Form.

Embolism (due to vegetation of the valve of the right heart).—Coagula from the right heart or some superficial vein.—Pyæmia (metastatic).—Phthisis.—Cholera.—Scurvy.

Causes of the Diffuse Form.

Aneurism.—Degeneration of the arterial coats.—Traumatism.

SYMPTOMS.

Rational.

Dyspnœa—if the apoplexy be extensive.

Sense of constriction in the chest.

Pain—if the pleura be affected.

Expectoration—of small blood-coagula.

If pyœmic in origin, the attack is accompanied by a chill and a marked rise in temperature.

Physical.

Percussion—local dullness over the seat of the disease.

Auscultation—respiratory murmur may be absent, if the air-celis are extensively involved.

Bronchial breathing is often detected over the seat of the disease.

DIFFERENTIAL DIAGNOSIS.

From pulmonary congestion; pulmonary œdema; and bronchial hemorrhage.

PROGNOSIS.

Is good, in the case of infarction, if not too extensive, and not dependent upon pyæmia or in connection with thrombosis.

Is bad, in the diffused form, unless slight in amount and of traumatic origin.

TREATMENT.

The patient must be kept in bed, and perfectly quiet. Stimulating applications, such as *mustard sinapisms*, must be applied to the extremities, and *dry cupping* to the chest-surface. Should it result from heart disease, regulate the heart's action, and increase its force by moderate use of *stimulants* and full doses of *digitalis*. If it occur with pyæmia, support your patient to the utmost, by giving *stimulants*, *quinine*, and *iron*, and apply dry cups over the chest-surface frequently. The diffuse variety is not amenable to treatment.

BRONCHIAL HEMORRHAGE.

DEFINITION.

Is a hemorrhage of the bronchial mucous membrane, and not of the parenchyma of the lung.

MORBID ANATOMY.

The bronchial mucous membrane, at the seat of hemorrhage, will be found to be swollen, relaxed, and bleeds on slight pressure. It is uniformly dark-red in color, with spots of ecchymosis, provided the examination be made soon after, or at the time of a hemorrhage. But should the examination be made some time after the bleeding, the bronchial mucous membrane will be pale, and bloodless in appearance, or, the seat of the hemorrhage will not be able to be discovered. If any of the small bronchi are occluded by blood-clots, the lung-tissue beyond the seat of the plugs will remain inflated after the thoracic cavity is opened.

ETIOLOGY.

Predisposing Causes.

Previously weak pulmonary capillaries are often a predisposing cause, as occurs:

In delicate persons of phthisical parentage.—In developed phthisis or a phthisical diathesis. — In chronic bronchial catarrh, which tends to weaken the vessels.

Exciting Causes.

Excessive exertion.—Atmospheric conditions.—Inhalation of irritant gases, etc.—Excessive rarefaction of air.

SYMPTOMS.

Rational.

Expectoration—is of bright arterial blood, with a frothy appearance.
Previous sense of constriction in the chest.
Cough and frequent return of hemorrhage.
Pallor and anxiety of face.
Tremulousness.
Syncope—is possible.
Nausea and vomiting—may follow the hemorrhage, but *never precede it.*
Symptoms of a subsequent pneumonia or phthisis may develop.

Physical.

Auscultation—large and small bronchial râles, over the seat of hemorrhage, may be detected.

DIFFERENTIAL DIAGNOSIS.

From gastric hemorrhage, thoracic aneurism, epistaxis, and pulmonary apoplexy.

PROGNOSIS.

Is seldom fatal, even in severe attacks, unless associated with some other disease. It is either the precursor of phthisical development, or a sign that phthisis exists.

TREATMENT.

The patient should be placed in bed, and kept absolutely quiet; not even being allowed to sit up, or move, or speak above a whisper. The room should be cool. *Opium* should be given in full doses, if the cough be constant and produce hemorrhage. In order to quiet the anxiety of the patient or friends, *ergot, spirits of turpentine, persulphate of iron*, or *chloride of sodium* may be given. *Ice bags* may be applied, but great care should be used in applying them, as broncho-pneumonia is apt to follow such attacks of hemorrhage. *Ice* should be eaten, and cold iced-water drank freely. The nutrition of the patient should be improved by *iron*, and a very nutritious but not stimulating diet. Avoid all mental

and physical exercise, but the patient should spend some hours daily in the fresh air.

PULMONARY GANGRENE.

DEFINITION.

A death of lung-tissue in mass—as contrasted with molecular death.

VARIETIES.

Circumscribed—where only small localized spots are affected.
Diffused—where extensive portions of the lung-tissue are involved.

MORBID ANATOMY.

In the *circumscribed form*, it usually occurs in the lower lobes or on the surface of the lungs. Small portions of the lung become converted into sloughs, which may be discharged through a bronchus and leave a ragged cavity surrounded by inflamed lung-tissue.

An opening is sometimes found in the pleural cavity, causing acute pleurisy, or hydro-pneumo-thorax.

In the *diffused form*, an entire lobe may be involved or even the entire lung; the gangrenous processes are not limited, but may become œdematous, or hepatized lung-tissue may be found. It may become converted into a putrid mass, and as the gangrenous process reaches the pleura, that membrane becomes destroyed, and the patient dies of pyæmia or septicæmia.

ETIOLOGY.

It may occur from:

Local Causes.

Pneumonia.—Hydatids.—Infarction (hemorrhagic).—Cancer.—Bronchial dilatations.—Embolism of an artery.

Nervous Diseases.

Dementia.—Cerebral softening.—Epilepsy.—Chronic alcoholism.

Blood-Poisoning.

Pyæmia.—Septicæmia.—Glanders.—Fevers.

SYMPTOMS.

Rational.

Marked fetor to the breath.
Expectoration—of a gangrenous material, or black lumps of lung-tissue.
Hemorrhage—in some cases.
Great physical prostration—(if septic poisoning occur).

Physical.

These are often obscure, and comprise those of consolidation, and, possibly, the existence of a cavity, viz.:
Percussion—localized dullness.
Auscultation — bronchial respiration; amphoric or cavernous breathing; and, possibly, gurgling.

DIFFERENTIAL DIAGNOSIS.

From abscess of the lung; phthisis; and fœtid bronchitis.

TREATMENT.

Sustain the patient with *stimulants*, tonics, and a most nutritious diet. Give *opium* to relieve pain, allay the cough, and overcome constitutional irritation.

CANCER OF THE LUNGS.

VARIETIES.

Medullary (as the rule); and melanotic, in rare cases. This disease may also be unilateral; or diffused, as nodules, throughout both lungs.

MORBID ANATOMY.

The medullary cancer is the only one met with in this disease, as a rule. Primary cancer only affects one lung, while the secondary form usually affects both. It occurs in the form of nodules scattered through the lung-substance. The nodules may blend and involve the entire lung. The cancerous matter may undergo fatty degeneration, and softening; it may also involve the pulmonary vessels and bronchi, or cause obliteration of them

through pressure. There are usually extensive pleuritic thickenings and adhesions present.

ETIOLOGY.

Predisposing Causes.

Hereditary tendency.—Is most common in the male sex, and occurs between 40 and 60 years of age.

Exciting Causes.

Cancer in the breast in females.—Injury.—Previous disease.

SYMPTOMS.

Rational.

Pain—in the chest.
Cough.
Expectoration—often like "currant jelly."
Dyspnœa—most marked when the mediastina are affected.
Emaciation and œdema of the face.
Temperature—elevated.
Night-sweats and exhaustion.
Hemorrhage—severe in some cases.
Cyanosis.
Vertigo.
Swollen jugulars.
Cachexia.—(may exist in some cases).

Physical.

Inspection—enlargement of the affected side, if the disease be extensively developed. Diminished respiratory movements.

Palpation—diminution, or absence of vocal fremitus.

Percussion—marked dullness over the seat of the disease with spots of resonance.

Auscultation—feeble or absent respiratory sounds. Bronchial breathing (if the cancer and a large bronchus be intimately connected).

DIFFERENTIAL DIAGNOSIS.

From pleurisy with serous effusion; gangrene of the lung; aneurism of the aorta; phthisis; or fibrous pneumonia.

PROGNOSIS.

Always fatal. Death may occur from exhaustion; hemorrhage; pulmonary œdema; asphyxia; or pressure on the œsophagus, trachea, or thoracic duct.

TREATMENT.

Is altogether palliative. The symptoms must be relieved as they arise.

PNEUMONIA.

DEFINITION.

Is an inflammation of the air-cells and intercellular passages—or, of the parenchyma of the lung.

VARIETIES.

Croupous or "lobar"—where a distinct lobe is affected.

Catarrhal or "lobular" or "broncho-pneumonia"—where a lobule is affected in different parts of the lung.

Fibrous or "interstitial" or "pulmonary cirrhosis"—where new connective-tissue development is the principal pathological change.

CROUPOUS PNEUMONIA (Lobar).

STAGES.

First, engorgement; *second*, red hepatization; *third*, gray hepatization.

MORBID ANATOMY.

In the first stage—the lung in the affected part is redder, and crepitates less than normal. The specific gravity is increased. The lung is firmer on pressure than normal. *On section*, you will perceive a reddish fluid, partly serous and partly blood, to exude from the cut surface. The *microscope* will show a congestion of the capillaries of the walls of the air-cells.

In the second stage—the affected lobe will be solid to the touch, and all crepitation absent. It pits on pressure and becomes

friable, and is of a dark-brown or reddish color; is heavier than normal, and sinks in water. *On section*, it will be granular (due to filling up of the cells with pneumonic material), giving it an appearance similiar to liver-tissue. About twelve hours after death, you get the post-mortem fluid, which, *under the microscope*, will be found to consist of fibrillated fibrin, blood-globules, lymphoid cells and large nucleated cells. The lung-substance will have lost the outline of the air-cells, if viewed microscopically.

In the third stage—the lung will be found mottled with dark spots, becoming uniformly gray (due to decoloration of melanine). On squeezing, it will now begin to crepitate, and has lost its solidity. The specific gravity is nearly normal, and the lung will sometimes float in water. *On section and squeezing* the lung, a milky or purulent fluid will be obtained.

Microscope.—The *outline of the cell wall* will be seen (from loosening and separation of the inflammatory material); and a granular substance in the centre of the cell will be perceived (which is a degeneration of the fibrin and the other inflammatory products). The blood-vessels will appear normal, and the epithelial lining will probably be intact.

ETIOLOGY.

Predisposing Causes.

Age (most common between 20 and 40).—Sudden change in temperature.—Poverty.—Intemperance.—Previous attacks.

Exciting Causes.

Exposure to cold or wet (as chilling of the feet or body).—Special diseases (as exanthematous fevers, uræmia, pyæmia, septicæmia, alcoholism, etc).—Traumatism.

SYMPTOMS.

Rational.

Chill—usually severe, prolonged, and distinct.
Headache, or vomiting, or bronchial hemorrhage.
Convulsions—(in children).
Coma—(in the aged).
Pain—(in the affected side for about three days).

Cough—(a constant symptom).
Expectoration—scanty, pinkish, and viscid (at onset); (later on), brick-dust, and tenacious; (severe case) resembling prune juice.
Temperature—elevated (highest on the third day, 102°-105).
Respiration—increased (80 per minute), panting in character.
Pulse—elevated (90 to 120)—is intermittent, in old age.
Countenance—anxious; flushed spot on the cheek; mahogany color (in severe cases).
Delirium—(in alcoholismus).
Urine—scanty, high-colored, high specific gravity, and albuminous.

PHYSICAL.

Stage of Engorgement.

Inspection—slight impairment of motion on the affected side.
Palpation—slight increase of vocal fremitus.
Percussion—slight dullness.
Auscultation—crepitant râle, (at the end of inspiration).

In Red Hepatization.

Inspection—marked impairment of motion on the affected side, and increased motion on the healthy side.
Palpation—marked *increase* of vocal fremitus.
Percussion—marked dullness.
Auscultation—bronchial respiration; bronchophony; increased intensity of vocal sounds.

In Gray Hepatization.

Inspection—partial return of motion on the affected side.
Palpation—slight increase of vocal fremitus and resonance over normal standard.
Percussion—decrease in dullness.
Auscultation—rude respiration or "broncho-vesicular;" *râle redux*, or modified subcrepitant, and crepitant.

DIFFERENTIAL DIAGNOSIS.

From cerebral disease, and cerebro-spinal meningitis (in children); from pleurisy in all its forms; phthisis (second stage); from capillary bronchitis; pulmonary œdema, and abscess of the liver

PROGNOSIS.

Is bad in infancy; old age; chronic alcoholism; in complications (as Bright's, heart, pregnancy); when very high temperature exists; if the pulse be over 150; in case of delirium after first week: if pulmonary œdema be present or double pneumonia, also when jaundice and prune-juice expectoration exist.

CAUSES OF DEATH.

Heart's failure and heart-clot; pulmonary œdema; asphyxia; and exhaustion.

TREATMENT.

Bear in mind the constitutional condition of the patient and trust Nature to help you. Blood-letting is only to be resorted to if the heart is engorged, and pulmonary congestion and œdema seem likely to take place. *Digitalis* or *nux vomica* should be given as a heart-tonic. Keep the patient in bed in a large, well-ventilated room, with a temperature about 68° to 70°, and give good nourishment (as milk, eggs, etc.).

Inject morphine hypodermically, if there is pain. Give *opium* or *chloral*, in small doses, should there be vomiting.

The dangers of this disease, in delicate subjects, are high temperature and feeble heart-power; it will therefore be advisable to reduce the temperature by giving *sulphate of quinine*, gr. xx.–xxx. per diem, and sustain the heart-power with alcoholic stimulants (judiciously applied—that is, just sufficient to tide the heart over the crisis).

If the pulse be rapid and feeble (120 to 130 per minute), *stimulants* should be given in small doses, not exceeding *six to eight ounces in the twenty-four hours. Belladonna* may be administered if the pupils are contracted.

Give *chloral*, in small doses, if the patient be restless and cannot sleep. Use *champagne* as a heart-stimulant. In convalescence, give *iron, oleum morrhuæ*, and a good nutritious diet.

CATARRHAL PNEUMONIA (LOBULAR) OR BRONCHO-PNEUMONIA.

DEFINITION.

The form which affects the scattered lobules and not a lobe of lung. Is most extensively developed in the anterior and posterior portion of each lung.

MORBID ANATOMY.

When the lung is inflated, there are small yellow or red spots which do not inflate, and which are not granular on section. If these spots be near the surface of the lung, they will be found to be rounded masses, and may be mistaken for tubercles. If the nodules are large, a reddish or grayish fluid exudes when cut; the nodules break down easily, and sink when thrown into water.

In the second stage or "stage of red hepatization," the exudation is made up of granular fibrin, blood-globules, lymphoid cells, and large nucleated cells.

In the third stage or "stage of gray hepatization," the nodules have a tendency to terminate either in *resolution*, *purulent infiltration*, or *cheesy degeneration*. The small bronchi are filled with tenacious mucus or muco-pus; their walls are thickened; and their calibre is enlarged.

If resolution or purulent infiltration occur, the inflammatory products undergo changes similar to those which take place in croupous pneumonia. If the products in the alveoli or bronchi become cheesy, a hyperplasia of the connective-tissue takes place, producing more or less induration (thus corresponding to catarrhal phthisis).

STAGES.

The same three as in croupous pneumonia, viz.: engorgement; red hepatization; and gray hepatization.

ETIOLOGY.

Predisposing Causes.

Most frequent in children, and follows pulmonary collapse; measles; diphtheria; whooping cough; bronchitis; prolonged recumbent position; or debility.

Exciting Causes.

Inhalation of an impure or irritating atmosphere.

SYMPTOMS.

Rational.

Previous symptoms of a bronchitis.
Temperature—rising to 104° or even higher.
Pulse and respiration—accelerated.
Respiration—becomes panting aud rapid.
Face—flushed and anxious.
Cough—dry, hacking, and painful.
Expectoration—scanty ; often absent ; seldom brick-dust in color.

In Acute Cases.

Lividity.—Extreme dyspnœa.—Convulsions.—Speedy death.

In Subacute Form.—(principally in strumous children).

Cough—severe and metallic.—Emaciation marked.—Marked pallor of face.—Night-sweats.—Gradual exhaustion.—Abscesses often form on nates and back.

Physical.

Percussion—slight dullness, in front and behind, (if pneumonic nodules are large).

Palpation—vocal fremitus slightly increased.

Auscultation—respiratory sounds feeble (when the tubes are obstructed); and bronchial in character, within the spots of consolidation. Crackling râles, of a fine and metallic quality, may often be detected after violent coughing, at the end of inspiration.

DIFFERENTIAL DIAGNOSIS.

From capillary bronchitis; collapse of the lung; croupous pneumonia; and acute tuberculosis.

PROGNOSIS.

Is bad, if secondary to scarlet fever, kidney disease, or infarction. Also bad, if developed in the weak; or if the temperature exceed 105°; or if due to pyæmia, or bad hygienic surroundings.

TREATMENT.

The patient should be kept in bed, in a large, well-ventilated room with a temperature about 65° to 70°, and the chest protected with flannel. Avoid all antiphlogistic measures or depressing remedies. *Stimulants* should be given *from the onset*, and continued to the end of the disease. *Dry cupping* should be the only counter-irritant used. *Quinine* should be administered (from gr. x.-xx. may be given daily to a child of three years of age). In convalescence, give *iron*, *oleum morrhuæ*, and good nutritious food. Avoid fatigue and exposure, and insist upon change of air.

FIBROUS PNEUMONIA.

SYNONYMS.

Interstitial, or "pulmonary cirrhosis."

DEFINITION.

Is that form of pneumonia associated with connective-tissue growth, and its subsequent organization and contraction. Is commonly called "chronic pneumonia."

MORBID ANATOMY.

There is, at first, a hyperæmia and swelling of the intercellular and interlobular tissue; followed by a rapid hyperplasia of the connective-tissue, which contracts, and therefore diminishes the size of the lungs. The chest-walls contract, in consequence of the diminution in the size of the lung; and the bronchi become dilated, owing to weakness and loss of their elastic power.

ETIOLOGY.

The predisposing causes are: encapsulated infarction; encapsu-

lated abscess; acute pneumonia; and splenization. It may occur as a result of phthisis.

SYMPTOMS.

Rational.

Retraction of the chest-wall over the affected lung.
Pain—in the affected side.
Cough.
Expectoration—profuse—if the bronchi are dilated—is often fœtid and contains cheesy matter.
Dyspnœa—not severe, although constant.
Emaciation.
Night-sweats.
Anæmia.

Physical.

Inspection—retraction of the chest-wall and loss of expansive motion.
Palpation.—the heart is often displaced toward the affected side. and signs of hypertrophy of the right side exist, due to obstruction of the pulmonary circulation.
Percussion—wooden; "cracked-pot resonance," over the dilated bronchi may be present.
Auscultation — feeble and bronchial breathing. Respiration—amphoric; or cavernous, if bronchi are dilated. Mucous râles —possibly gurgling in character.

DIFFERENTIAL DIAGNOSIS.

From pleurisy with effusion; pleurisy with retraction; and cancer of the lung.

PROGNOSIS.

Is good, as to duration of life; but bad, if complicated with diarrhœa, hemorrhage, or general dropsy, from either heart or kidney lesions.

TREATMENT.

This disease, when fully developed, is incurable. If it be due to a bronchitis, it is advisable to guard against its recurrence. In

order to get rid of the accumulated secretion, and prevent a fœtid accumulation, inhalation of benzoin or oil of turpentine has been recommended.

PLEURISY.

DEFINITION.

Is a partial or general inflammatory exudation into one or both pleural cavities.

VARIETIES.

Acute pleurisy—is well defined; runs a rapid course; and has a *fibrinous* exudation.

Subacute pleurisy—is mild; of slow development; and has a *serous* exudation.

Chronic pleurisy, or "*empyema*"—is of slow development; has a *sero-purulent* exudation; and occurs in persons of debilitated constitutions.

Hydro-pneumo-thorax—is a modification of the chronic form, with *perforation of the lung*, admitting air into the pleural cavity (in which fluid also develops, later on).

ACUTE PLEURISY.

MORBID ANATOMY.

There will be a reddening of the pleural surface, extending to the lungs or costal tissues; and, possibly, ecchymosis. New connective-tissue is formed; with new vessels which have thin walls. The pleura will have a thick, swollen, rough, and shaggy appearance, and have lost its lustre. Its surface may be agglutinated by coagulable lymph (fibrin), causing connective-tissue adhesions. These adhesions are covered by a layer of pavement epithelium, within which run long slender blood-vessels. These adhesions may be permanent; or contract, and then undergo fatty degeneration and absorption, thus producing a simple thickening of the pleura.

ETIOLOGY.

It may occur as a primary or secondary disease, and at any age.

Primary form may follow

Exposure to cold or dampness.—Injury.

Secondary form may be due to

The different fevers.—Rheumatism.—Bright's disease.—Pyæmia.—Septicæmia.—Alcoholism.—Pneumonia.—Cancer of the lung.—Phthisis.

SYMPTOMS.

Rational.

Localized pain—in the side, *usually near the nipple.*

Increase of pain on inspiration; respiration "catching."

Bending of the trunk toward the affected side.

Countenance—pale and anxious; may become flushed as the disease advances.

Pulse—increased (90 to 120), firm, small and tense.

Temperature—is seldom above 100°—may reach 104°.

Cough—hacking; usually no expectoration is present.

Thirst.

Tongue—usually coated.

Nausea and Vomiting—occasionally present in the early stages.

Dyspnœa—sudden (if the pleural cavity be over-distended).

Chills—and, possibly, expectoration of pus (if empyema is developed).

Physical.

Inspection—restricted movement of the affected side.

Palpation—vocal fremitus absent, below the level of the fluid.

Percussion—dullness, till the fluid accumulates. Flatness below the line of the fluid (the line of which *changes with the attitude* of the patient).

Auscultation — friction sounds are heard (before the fluid is formed). Vocal sounds are absent, below the level of the fluid (after it is exuded). Bronchial breathing, below the level of the fluid (in exceptional cases). Return of friction and voice sounds (as the fluid is absorbed).

Retraction of the chest-wall often follows this disease.

DIFFERENTIAL DIAGNOSIS.

From intercostal neuralgia; pneumonia; pulmonary œdema; phthisical consolidation; thoracic aneurism.

PROGNOSIS.

Is good, if of the primary form; grave, if following any severe form of disease, or if absorption be too long delayed; also if empyema be developed; or if phthisis be a complication.

TREATMENT.

Inject opium, hypodermically, to relieve pain. Patient must be kept quiet in bed, in a large, well-ventilated room, at a temperature about 65° to 70°, and have good nourishing diet without stimulants.

Give *syr. ferri iodidi*, ʒ i. three or four times daily, if there is anæmia.

SUBACUTE PLEURISY.

SYNONYM.

"Sero-fibrous" pleurisy.

MORBID ANATOMY.

There is more extensive connective-tissue formation than in the acute form; the pleura is more thickened and there is more effusion (serous). The serous fluid in the pleural cavity does not degenerate into pus; but an abundant cell-formation takes place. The intercostal spaces are distended; the diaphragm is pushed down; the viscera are displaced; and the lung is pushed upward and inward against the spinal column (if there is much effusion), or compressed (becoming of a pale-red or green color, tough and leathery, and containing no air). As the fluid disappears, adhesions form; the pleura becomes thickened; the chest-walls retracted; the lungs inexpansible; and the liver may rise higher than normal.

ETIOLOGY.

The same as in the acute form: it is also secondary to Bright's,

phthisis, etc., and occurs in the week and feeble. It is often the first stage in the development of phthisis.

SYMPTOMS.

Rational.

Pain—is rare (but there is, often, a sense of uneasiness after exercise).
Emaciation.
Dyspnœa—on exertion.
Cough.
Expectoration—muco-purulent, or mucus.
Pulse—small and feeble (110 to 120).
Dryness of the skin—(especially at night).
Temperature—about 101°.
Countenance—pale and anxious.
Breathing—short and catching (patient *can only lie on the affected side*).
In the active form.

The symptoms are active. They will increase for a time; then cease; and subsequently return with greater severity. There is a rapid increase of fluid, causing intense dyspnœa and cyanosis, and œdema of the other side (from the pressure created). Patient is frequently unable to lie down.

Physical.

If *the cavity be partly or completely filled.*

Inspection—the affected side is enlarged in all directions. *Bulging* of the intercostal spaces may be detected. Respiratory movements on the affected side are upward and downward, and are increased laterally on the other side. There may be entire absence of respiratory movements on the affected side. The heart is displaced; a fullness, below the ribs, in the abdomen may exist (from the liver being pushed down below the free border of the ribs).

Mensuration—the affected side is larger than normal after expiration, (one or two inches).

Palpation—complete absence of vocal fremitus.

Percussion—flatness of the affected side (below the level of the fluid).

Auscultation—entire absence of the respiratory and vocal sounds (below the level of the fluid). *Bronchial respiration* and *bronchophony* above the fluid (if the lung be sufficiently compressed).

As absorption takes place, the vocal sounds return to normal, and a creaking, rubbing, friction sound is produced as the two surfaces play on each other. The surfaces of the pleura are thickened (due to new connective-tissue formations). The vocal fremitus is diminished; the respiration is feeble; and there is *dullness* on percussion, with a tendency to return to a vesicular quality, as the lung expands.

DIFFERENTIAL DIAGNOSIS.

From pneumonia; consolidation of the lung; enlarged spleen and liver; cancer of the lung and pleura; and aneurism.

From pneumonic and phthisical consolidation of the lung.

There is the history to aid in the discrimination.

The *expectoration* of pneumonia is pink, brick-dust, or "prune-juice."

The expectoration of phthisis is *streaked* with blood.

Percussion—there is dullness, but not a flatness which changes with the position of the patient (as in pleurisy).

Auscultation—vocal fremitus is intensified and increased; the respiration is feeble; bronchial and moist râles exist.

The symptoms of pulmonary consolidation begin from above; extend downward; and are often bilateral.

From the spleen (when enlarged).

The enlargement extends upward and downward; is confined to the abdominal cavity; and there is continuous dullness.

From cancer of the lung.

Aspiration with the hypodermic needle, and the microscope will settle the question; and the physical signs may assist in the diagnosis.

PROGNOSIS.

Is good, if there is no complication; but if there is over-distention from fluid and marked dyspnœa, the prognosis is bad.

TREATMENT.

Syrup of the iodide of iron (℥ i.), three or four times daily should be given; also a large amount of good nutritious food. The patient may be given a bottle of wine daily, and *the nutritive process should be raised to the highest possible degree;* as, by keeping up the vital powers to their utmost, you thus aid Nature to get rid of the fluid in any serous cavity. Cathartics, diuretics, diaphoretics, or blood-letting *should never be resorted to*, as there is too great an increase of fibrin, and they have no power to remove it.

Aspiration (see hydro-pneumo-thorax) should be resorted to early if the effusion is very great, until all the fluid, *necessary to relieve extreme pressure*, is removed; but great care should be used, as pulmonary emphysema or phthisis may ensue if carried too far. In the young, a systematic course of gymnastics may be resorted to in order to overcome the adhesions (should they occur).

CHRONIC PLEURISY.

SYNONYM.

Empyema.

DEFINITION.

There is pus, or a purulent exudation, in the cavity of the pleura; due to an inflammation, which may be primary or secondary, and which comes on gradually (in the majority of cases).

MORBID ANATOMY.

A pouring out of plastic material, which undergoes transformation into pus, occurs (especially in pyæmia and septicæmia); and a migration of white blood-corpuscles through the walls of the blood-vessels into the connective-tissue of the pleura, from which they are washed by the serous effusion, and held in suspension, may also take place in other conditions. The exudation is at first clear; but, later on, consists of yellow, greenish-yellow pus (primary empyema); this exudation goes on increasing, causing the

pleura to become a "pyogenic membrane" (produced by excessive irritation). If the irritation is mild, the cell-production may be circumscribed and not general. The accumulations may escape by spontaneous openings, such as perforation of the chest-walls by ulceration; perforation of the lung, and its escape by the bronchi; perforation through the diaphragm, entering the abdominal cavity, producing fatal peritonitis and death; or into the intestinal canal, through adhesions between the intestines and the diaphragm, when pus will appear in the discharges from the bowels. If recovery takes place, there is a general contraction, by cicatrization, of all the viscera, to fill up the gap and stop the purulent discharge.

ETIOLOGY.

Exciting Causes.

Traumatism; broken down subjects; phthisis; protracted exhaustive diseases (as advanced phthisis, suppuration of bones, chronic syphilis, chronic alcoholismus, cancer, or special vice).

SYMPTOMS.

Rational.

Pain—is rare.
Uneasiness and weight on the affected side.
Countenance—pale and anxious.
Hectic chill, fever and sweating (at intervals).
Cough.
Expectoration—muco-purulent, and not abundant.
Dyspnœa.
Development of a condition similar to that of phthisis.
Semi-comatose state—if pyæmia or septicæmia occur.
Protrusion of a tumor—between the ribs (which fluctuates and is red, if about to discharge externally).
Pneumonic signs—(if the discharge take place through the lungs) such as chill, cough, fever, profuse muco-purulent expectoration with blood, retraction of the chest-walls, etc.

Physical.

Same as those of the subacute form, excepting that the level of the fluid does not change so readily.

DIFFERENTIAL DIAGNOSIS.

Is impossible, except by aspiration. Is chiefly mistaken for pleurisy.

PROGNOSIS.

Is *bad.* It is stated, that one in every five recover from spontaneous openings; and one-eighth, where aspiration is resorted to. Death occurs from exhaustion.

TREATMENT.

Early aspiration. The patient should be placed upon the *most nutritious diet*, and only a small amount of stimulants given. *Iron, quinine, and cod-liver oil* are the best medicinal agents to rely upon. Let the patient be in the open air as much as is possible, or order a change of climate.

HYDRO-PNEUMO-THORAX.

DEFINITION.

Is a condition, characterized by the presence of fluid and air within the pleural cavity.

MORBID ANATOMY.

The changes are the same as in empyema. There may be a great quantity of air and small amount of fluid in the pleural cavity, or the reverse.

ETIOLOGY.

Traumatic perforation of the chest-wall; perforation of the lung by tubercle, cancer, etc.: traumatic rupture of an emphysematous lung; fracture of the ribs; or gangrene of the lungs.

SYMPTOMS.

Rational.

Dyspnœa—extreme (when the air first enters).
Pain—(intense and thoracic) of the affected side.
Cyanosis—very marked at onset.
Collapse and death—may occur from shock, but continued dys-

pnœa; filling up of the chest with fluid (usually pus); general dropsy; or hectic fever most often follow.

Symptoms of exhaustion or cyanosis (from pulmonary œdema).

PHYSICAL.

Inspection—bulging of the affected side; diminished movement of the chest.

Palpation—absence of vocal fremitus on the affected side.

Percussion—tympanitic, above the fluid; flatness, below the fluid.

Succussion—(a splashing sound heard on shaking the patient).

Auscultation—"metallic tinkle" (due to the dropping of fluid from the lung into the fluid below).—Amphoric respiration.

The *pathognomonic sign of fluid in the cavity* is *dullness on percussion, and alteration of the dullness*, due to the change in position of the patient. This fluid may, however, be held down by adhesions (or encapsulated), so that its position is not altered, in which case this sign will be absent.

DIFFERENTIAL DIAGNOSIS.

From pneumo-thorax; obstruction to a bronchus; emphysema (if extensive); or a large cavity in the lung.

PROGNOSIS.

Is unfavorable in every case; and rapidly fatal, if due to pulmonary gangrene or phthisis.

TREATMENT.

Is palliative. *Inject morphine* hypodermically. If the patient survive the first few days, give *stimulants* and good nutritious diet. *Paracentesis* should only be resorted to in extreme cases. *In performing aspiration*, place the arm of the affected side at right angles across the chest, and insert the canula (for a distance of two inches) at a point situated about the line of the sixth rib and at the junction of the infra-scapular and axillary regions; so soon as the patient complains of restriction, stop the operation; then wait for a day or two before performing it again. This operation should be persevered in until the fluid gets below the line of the scapula.

HYDRO-THORAX.

DEFINITION.

Is a non-inflammatory accumulation of fluid within the pleural cavity.

ETIOLOGY.

It occurs in general dropsy of Bright's disease; in the course of general hydræmia; or chronic exhaustive diseases; in valvular heart-lesions; or it may be due to mediastinal tumors.

SYMPTOMS.

Rational.

Dyspnœa—steady and progressive.
Cyanosis—increasing steadily.
Respiration—gasping (when the fluid is excessive).
Inability to lie down.
Pulse—feeble and small.
Temperature—not increased.
Articulation—monosyllabic (in advanced stages).

Physical.

Inspection—gradual impairment of motion of the chest, and bulging of the two sides.
Percussion—flatness, below the level of the fluid, which changes with the attitude of the patient.
Auscultation—absence of the vocal sounds, below the level of the fluid.

DIFFERENTIAL DIAGNOSIS.

From subacute pleurisy; and pulmonary œdema.

PROGNOSIS.

Is modified by the cause, and condition of the patient. It may prove a direct cause of death in Bright's disease, or when a chronic heart-affection exists.

TREATMENT.

The use of hydragogue cathartics and diuretics, and those agents which are employed to remove fluid from the areolar tissue, has been long in vogue; but *the best mode of treatment is the aspirator.*

PHTHISIS.

DEFINITION.

Is a "wasting away" or molecular death of the lung-substance.

VARIETIES AND SYNONYMS.

"CATARRHAL" OR "CHRONIC CATARRHAL PNEUMONIA"—where the disease is a *continuation of a pneumonia* (catarrhal type).

"FIBROUS" OR "CIRRHOSIS" OR "INDURATION"—where the *connective-tissue growth* is prominently developed.

"TUBERCULAR" OR "ACUTE MILIARY TUBERCULOSIS" — where *miliary tubercle* is deposited within the lung in the lymphatic structure.

MORBID ANATOMY.

CATARRHAL FORM.—There has been a previous *catarrhal* or *lobular pneumonia*, which has not been followed by *resolution* during the third stage; but whose *products have become caseous.* This caseous matter creates ulceration of the lung by acting as a foreign body, and, possibly induces miliary tuberculosis by absorption. In rare cases, this caseous matter may become encapsulated and innoxious.

FIBROUS FORM.—There is (1) proliferation of new connective-tissue cells in the lung; (2) organization of these cells and formation of new connective-tissue; (3) contraction of this new connective-tissue, creating pressure on the vesicular structure.

TUBERCULAR FORM.—There is absorption of caseous matter from some other part of the body resulting in lymphatic formation in the peri-vascular spaces of organs (especially of the lungs), which under the microscope shows giant cells. The tubercles subsequently undergo ulceration and excavation (probably by the

pressure exerted on the blood-vessels, causing impairment of nutrition).

ETIOLOGY.

Predisposing Causes.

Hereditary or acquired constitutional debility.—Bad hygiene.—Damp, cold climate.—Badly drained, or miasmatic soil.

Exciting Causes.

Extension of a bronchial catarrah.—Pneumonia.—Subacute pleurisy.—Inhalation of irritating particles or gases.—Exposure to cold, or sudden changes in temperature.

SYMPTOMS.

Rational.

Constant elevation of temperature.
Cough—dry, hacking or paroxysmal in early stages.
Expectoration—streaked with blood, often slight in amount (indicating lobular consolidation).
Hæmoptysis.
Emaciation—steady and gradual.
Night-sweats—commence early in the disease.
Dyspnœa—at first, only after exertion; but, later on, a constant symptom.
Pain in chest—due to local pleurisies.
Hectic fever—when cavities are formed.
Pulse—accelerated (100 to 140), corresponds to temperature.
Disturbance of digestive function.
Vomiting—usually due to reflex causes.
Diarrhœa (bad symptom)—due (1) to indigested food ; (2) follicular ulceration of the small intestines ; (3) ulceration of the large intestines (occurring usually in later stages)
Arrest of menstruation—(is often an early symptom).
Aphonia—partial (from ulceration of the larynx).
Œdema of legs and feet—(due to thrombosis in lower extremities).
Clubbing of the finger nails.
Fatty liver—(is possible), consists of simple deposit of fatty matter.
Face—peculiar clearness of the sclerotic.

Increased sexual desire.
Cyanosis (when the disease is extensive).

PHYSICAL.

Stage of Consolidation.

Inspection—slightly diminished expansion of the chest. Depression in the infra-clavicular spaces.

Palpation—vocal fremitus increased.

Percussion—Slight dullness (as compared with the other side).

Auscultation—Respiration, rude or bronchial. *Prolonged and high-pitched expiration.* Râles — subcrepitant, mucous, crepitant, or metallic. Vocal resonance intensified.

All of these symptoms of the first stage are important, as they often enable the practitioner to offer a favorable prognosis. They should be always determined by a *comparison between the two lungs.*

Stage of Softening.

Inspection—diminished expansion of the chest. Respiration more frequent. Marked depression above and below the clavicle.

Palpation—increased vocal fremitus.

Percussion—dullness increased (wooden).

Auscultation—bronchial breathing. Abundant râles, circumscribed and bubbling. Crackling sounds, at certain spots.

Stage of Excavation.

Inspection—retraction of chest-walls.

Percussion—Dull, amphoric, or cracked-pot.

Auscultation—Respiration, cavernous or bronchial, and gurgles. "Cavernous whisper."

DIFFERENTIAL DIAGNOSIS.

From bronchitis (all forms); croupous pneumonia, especially if at apex or accompanied by typhoid fever; pleurisy; pulmonary infarction and hæmoptysis.

PROGNOSIS.

In the catarrhal form, the disease may often be arrested, *in its first stage*, by change of climate.

If the tubercular variety exist, the prognosis is unfavorable.

If the fibrous form be developed, the prognosis is good as regards duration of life. If complications arise, as laryngitis, pulmonary œdema, capillary bronchitis, pulmonary congestion, or profuse hemorrhage, the prognosis is bad.

TREATMENT.

Is usually considered under three heads:

First.—The prophylactic treatment.

Second.—The medicinal treatment.

Third.—The hygienic treatment.

The most satisfactory results are usually obtained where the *prophylactic treatment* can be resorted to early; for, if the patient live in a district which subjects him to any form of depression, a change of residence should be insisted upon. A phthisical, or delicate mother should never be allowed to nurse her infant, but a wet-nurse should be procured. During childhood, the offspring should be fed on cow's milk and removed to the country, where it can have plenty of good fresh air, and physical training. Those who are predisposed to phthisis should never be allowed to breathe an atmosphere laden with dust or charged with poisonous vapors; neither should they be exposed to sudden changes in temperature. This class of patients should always sleep in large and well-ventilated rooms, and free from overcrowding. Physical exertion, such as violent running, jumping, etc., should be avoided. The surface of the body should be always covered with flannel. The diet should be simple, and taken at regular intervals, and consist of such articles as the stomach is able to digest without any sensation of weight or pain. *Stimulants* should *only* be resorted to in case of extreme fatigue or severe exposure to cold, as the constant use of them is injurious. This class of patients is very susceptible to catarrh on the slightest exposure, and these attacks should be treated with the greatest care; but, if it shall have reached the smaller bronchi, there is nothing so certain to remove the catarrh as a change in climate. In order to carry out this form of treatment most effectively, the vital powers of the patient should be sustained, so that he should not become susceptible to the local causes of phthisis.

The Medicinal Treatment.—The worst symptom to be encountered in this disease is *fever*. By the rise in temperature, it is evident that a bronchitis has resolved itself into a broncho-pneumonia; by a still greater rise, the products are becoming cheesy; and by the hectic fever and night-sweats, softening is commencing. It is therefore essential that the temperature should be at once reduced, and the only drug which can be relied upon is *sulphate of quinine*. This drug has also been regarded as a powerful agent in the relief of night-sweats; there is no doubt that it has this power, but it must be borne in mind that night-sweats are only a part of the fever, and that in controlling the one you control the other. When the first elevation of temperature occurs, *quinine* (gr. xx. per diem, given in one or two doses), rarely fails to control the temperature, diminish the frequency of the pulse, and arrest symptoms which are liable to result from the fever. It will be well to determine the time at which the highest temperature occurs and administer a large dose one or two hours preceding the attack, and continue it for as long a period as possible without producing cinchonism or until the temperature falls. *Cod-liver oil* is another medicinal agent. If possible, it should be given with an alkali in small doses, and not often repeated. A teaspoonful once or twice a day is sufficient to commence with. It may be gradually increased to a tablespoonful three times a day. Avoid administering it in connection with stimulants, unless the patient cannot take it in any other way. There are three forms which afford some clue to the mode of its action. First, unless the patient gains weight while using it, it seldom if ever proves remedial; secondly, flesh and weight may be gained during its administration and still the phthisical processes steadily progress; thirdly, when it does act remedially, the weight gained is far greater than would result from the oil as a mere element of nutrition. The *hypophosphates of lime* are only serviceable when intestinal digestion is imperfect. *Iron* is only of service when there is no marked fever; since if administered when there is fever the digestion will be disturbed and diarrhœa probably ensue. *Stimulants* may be given when they increase the desire for food and assist digestion, or when their use is followed

by a feeling of increased strength and a disposition to take exercise; but if they produce symptoms of fever and depression they will do harm. *Opium* should never be given in any stage of phthisis unless the cough is distressing and the patient is unable to obtain the requisite amount of sleep; under such circumstances the inhalation of a few drops of *chloroform* is preferable to opium. Vomiting after meals is often a troublesome attendant of this disease. The best means to be adopted in this case is to compel the patient to take, every half-hour for forty-eight hours, about a teaspoonful of *raw scraped beef* made into a sandwich, at the same time keeping absolutely quiet in a recumbent position.

The Hygienic Treatment.—This mode of treatment is by far the most important to phthisical subjects. It will be well to bear in mind the following rules, which will greatly assist in alleviating the sufferings of the patient:

1. It is absolutely necessary that there should be a good supply of pure and fresh air.

2. Exercise in the open air should be resorted to daily, when practicable.

3. The climate in which such patients should live, must be uniform, sheltered, temperate, and mild (about 60°), with a range of not more than 10° to 15° during the year. A *cold, dry climate* often agrees with some patients of this class; its beneficial effect can be estimated by the relief afforded to the cough and a tendency to produce healthy sleep. The soil should be dry, and the drinking water pure and not hard.

4. The dress should be of such a character as to equalize and retain the temperature of the body.

5. Late hours should be particularly avoided, as also indoor and sedentary occupation.

6. Cleanliness, which is reputed to be next to godliness, should be scrupulously attended to.

7. The diet should be most nutritious, easy of digestion, and more or less varied; and the number of meals should not be restricted. Milk, eggs combined with milk, beef tea (as a concentrated nutrient), and koumiss (made from asses' milk), are highly recommended for this class of patients.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE PERICARDIUM.

The pericardium is composed of two layers (a fibrous and serous). The fibrous layer is closely attached to the diaphragm: the serous closely adheres to the internal surface of the fibrous layer, is reflected from the large vessels, and completely invests the heart.

ACUTE PERICARDITIS.

DEFINITION.

Is an inflammation of the covering membrane of the heart

MORBID ANATOMY.

The serous surface is more or less reddened with ecchymotic spots; the reddening may be circumscribed or diffused; and the discoloration is due to hyperæmia of the sub-serous capillary vessels. There is also swelling and infiltration of the sub-serous tissue ; while the epithelial covering is separated and thrown off, thus losing its natural, brilliant appearance. If the inflammation is continued, an exudation is poured out on to the free pericardial surface.

The plastic exudations vary in thickness, accumulating on the cardiac and parietal surfaces, or the cardiac surface alone. As soon as the plastic material is poured out, it causes roughening of the pericardium (commonly called "hairy heart"). The fluid may be sero-albuminous, sero-fibrinous, hemorrhagic, or purulent. There may often be from three to twelve fluid ounces in the sac (which is usually sero-fibrinous in character); sero-albuminous fluid is very rare. If the pericardium is filled with fluid, the lung becomes compressed. The effusions may undergo absorption, or remain as cheesy masses. Bands, or adhesions may form, or there may be agglutination of the surfaces and obliteration of the

pericardial cavity. Myocarditis may be produced by extensive and long-continued pericarditis, producing weakness of the walls of the heart. Dilatation of the cavities may also take place through the weakened condition of the heart-walls, and hypertrophy may be developed as a result of this weakening, or as a secondary result of dilatation.

Post-mortem shows, white spots on the external surface of the heart, caused by a growth of connective-tissue beneath the cardiac pericardium; this is really a localized pericardial inflammation without adhesions. The two surfaces of the pericardium may also be found to be agglutinated, causing obliteration of the pericardial cavity.

ETIOLOGY.

It is always a secondary disease, and occurs chiefly in young people. It may arise from *inflammation* of the neighboring organs, as in pneumonia; pleurisy; neurosis of the sternum and the ribs; and from certain *blood-diseases*, as acute rheumatism, Bright's disease, scarlatina, small-pox, typhus, tuberculosis, syphilis, alcoholism, scurvy, purpura, etc.

SYMPTOMS.

Rational.

It comes on insidiously, as a rule, on account of its frequent complication with other diseases.

Pain in the precordial space—slight or lancinating, involving the brachial plexus, extending down the arm, and increased by pressing up the diaphragm.

Cough—dry, irritating, and a sense of constriction over the chest.

Dyspnœa—depends upon the amount of fluid effusion present.

Countenance—anxious.

Restlessness—if the effusion be considerable in amount.

Posture—half sitting, and leaning towards the affected side; or on the back with the head elevated.

Pulse—(before fluid effusion) full and strong (90 to 120); (after fluid effusion) feeble, suppressed, delayed, or intermittent.

Temperature—usually, about 100°; sometimes below normal, which is a serious symptom.

Headache—frequent dizziness.
Delirium—active (occasionally).
Syncope—on moving, if the fluid be extensive. This is probably a symptom of myocarditis.
Heart's action—forcible and irritable. There is an *increased area* of apex-beat.
Palpitation—(due to compression of the heart).

PHYSICAL.

Inspection—diminished respiratory movements, over the pericardial space.—If the pericardial sac be distended, there is arching of the precordial region from the second to the sixth intercostal spaces.

Palpation—the apex-beat is raised and carried to the left.—*If effusion* exist—cardiac excitement and friction fremitus disappear; the apex-beat is feeble, or imperceptible; and an undulating impulse is felt.

Percussion—If effusion exist—dullness is increased laterally and vertically. — *Laterally* — from one nipple to the other (in marked cases).—*Upward*—as high as the second or first rib. *Downward*—more than normal (displacing the diaphragm).

During stage of Plastic Effusion.

Auscultation.—Pericardial friction sounds, may be single or double, and independent of the heart-sounds; are superficial, and may, or may not accompany the heart-sound; the maximum of intensity is at the junction of the fourth rib and the sternum on the left side.—The intensity of the friction sounds increases by change in position, and also by a full inspiration (due to pressing of the two pericardial surfaces together by the distended lung).

During Stage of Fluid Effusion.

An absence of respiratory murmur may exist (from the lung being pushed to the right and left, by the distended pericardial sac), and the heart's sound may be rendered feeble or be entirely lost.

Stage of Absorption.

On recovery taking place, the bulging subsides; the dullness decreases; the sufaces come together and often adhere

(due to the inflammatory process); the friction sounds reappear; the heart-sounds are more distinct; the apex-beat becomes normal; the cardiac impulse returns; and the respiratory and vocal sounds are again detected.

DIFFERENTIAL DIAGNOSIS.

Between pericardial friction sounds, and endocardial murmurs.
Between pericardial friction sounds, and pleuritic friction sounds.
Between pericardial effusion, and hypertrophy and dilatation of the right ventricle.

PROGNOSIS.

There is generally complete recovery, except when it occurs in connection with Bright's disease, and septicæmic and pyæmic conditions. If pyæmia exist, you have a purulent exudation which is rarely absorbed. The nature of the exudations determine the prognosis. Acute pericarditis may become chronic, or be accompanied by large serous effusions which disappear slowly and may be accompanied by relapses. It may create extreme dyspnœa and, in rare cases, fatal syncope. If there is a long continuance of the fluid, the heart softens, and there is degeneration of its muscular tissue; this organ then becomes enfeebled, its force diminishes, and death ensues from œdema of the lungs, possibly associated with blood-changes, from loss of red blood-globules and fibrin. If it occur from acute rheumatism, it is rarely fatal; but you have as sequelæ, adhesions of the two surfaces, cardiac dilatation, and hypertrophy. This dilatation is due to a weakness of the walls of the heart from the inflammatory process; and the hypertrophy which follows the dilatation is compensatory. There are also further sequelæ, such as abundant exudations, and extensive adhesions at the base of the heart, causing contraction, pressure, and possible obstruction through the coronary arteries, producing fatty degeneration of that organ.

TREATMENT.

You must support your patient. First seek the cause and remove it. If it be due to fevers and the resulting depression, or to pyæmia, or septicæmia, give stimulants in moderation. In the

early stages of the disease, hot anodyne poultices (opium, belladonna, etc.) should be employed. Give opium internally in small doses; the largest doses may be given at night in order to produce a quiet sleep, but it should never be carried to narcotism. If there be effusion, it should be removed by medical measures, if possible. If you are able to sustain your patient, and there are no evidences of pulmonary congestion and œdema, immediate interference may be delayed. Do not employ hydragogue cathartics, blisters, etc., as the disease, in all cases, is due to some previous weakening disease, and consequently these remedies make it worse. Iron and stimulants should be used in moderation; also concentrated nutrition. The patient should be placed in bed and kept perfectly quiet. The chest must be protected from cold, and anything that accelerates the heart's action be avoided. In convalescence, the patient must be watched on account of the weakness of the heart's walls, and dilatation and hypertrophy that are present. If there be pus in the pericardium, then aspirate, and only for that condition.

CHRONIC PERICARDITIS.

MORBID ANATOMY.

In some cases, the pericardial sac contains fluid, adhesions, chalky debris, and calcareous plates.

ETIOLOGY.

If the acute form does not terminate in three or four weeks, it may be considered as of the chronic type.

SYMPTOMS.

Rational.

Obstructed circulation.
Enlarged heart (as shown by percussion and position of apex-beat).
Dyspnœa (from pressure on the lung and weak heart's action).
Sense of weight in the precordial region.
Angina pectoris—(in some instances).
Palpitation—on slight mental or physical exertion.

PHYSICAL.

Inspection.—Depression of the precordial region may occur (from firm adhesions taking place between the pericardium and the chest-walls).

Palpation.—Apex-beat is indistinct, and often two inches higher than normal. Displaced cardiac impulse, unattended by change of position of the organ. Irregular movement of heart, in systole and diastole.

Percussion.—Increased dullness, in the pericardial region.

PROGNOSIS.

Is doubtful; especially when degeneration of the cardiac walls and valvular insufficiency exist.

TREATMENT.

Limit the physical exercise so as not to overtax the heart. The diet must be most nutritious, but not stimulating; administer *iron.*

HYDRO-PERICARDIUM.

DEFINITION.

Is a non-inflammatory sero-albuminous exudation into the pericardial cavity, rarely causing death.

ETIOLOGY.

It occurs most often in renal diseases, which are liable to complicate scarlatina; also in chronic Bright's disease; general dropsy; and chronic cardiac disease, (result of venous congestion).

SYMPTOMS.

The same as those which occur in the stage of fluid effusion of pericarditis, except that there is no febrile disturbance, neither are there any friction sounds present at any time during the progress of the effusion.

PROGNOSIS.

Is a precursor of death in chronic Bright's, and advanced cardiac diseases.

TREATMENT.

The same as in hydro-thorax. Treat the disease that caused it.

PNEUMO-PERICARDIUM.

DEFINITION.

Air in the pericardium, due to compound fracture of the ribs, or external wounds, or, to the ulcerative process of phthisis.

DIAGNOSIS.

This rests almost exclusively upon the *tympanitic percussion* over the precordial space, and, the *tinkling* and *splashing sound* which is heard over the heart.

PROGNOSIS.

Is always fatal—except when due to traumatism.

HÆMO-PERICARDIUM.

ETIOLOGY.

Blood in the pericardial sac may be due to traumatism; rupture of the cardiac walls; or distention of the pericardium with blood from rupture of an aneurism, septicæmia, pyæmia, scurvy, purpura, etc.

PROGNOSIS

Unless traumatic, is rapidly fatal.

TUBERCULOSIS OF PERICARDIUM.

ETIOLOGY.

Is only met with in acute general tuberculosis, producing pericarditis. It may be suspected, if *general tuberculosis* be present.

CANCER OF THE PERICARDIUM.

ETIOLOGY.

This disease is always secondary to cancerous developments in other parts of the body.

ACUTE ENDOCARDITIS.

DEFINITION.

Is an inflammation of the (serous) lining membrane of the heart, from blood-changes, causing alterations especially in the neighborhood of the valves, and, possibly, involving the whole endocardial surface of a ventricle or auricle. Is most common in left heart.

VARIETIES.

Acute, which gradually runs into *chronic*, with no defined line of separation; except, that the acute symptoms last three or four weeks, and then the disease becomes chronic in character.

MORBID ANATOMY.

1st. There is hyperæmia of the endocardium, most marked about the edges of the valves.

2d. A plastic exudation on the free surface (which is washed off by blood), occurs at the same time; also a growth of new cells underneath the inflamed portion of the endocardium, which by their presence produce irregularities upon the free surface of the valves. The valves are also somewhat tumefied by inflammatory œdema.

3d. The elevations, on the free surfaces of the valves, produced by the new cell-growth underneath the endocardium, create a deposit upon them of the fibrin of the blood, resulting in the so-called "vegetations."

4th. The new cell-growth under the endocardium organizes into new connective-tissue; and this subsequently contracts, causing an imperfect performance of the function of the valves.

5th. The free edges of the valves sometimes become adherent to each other, producing stenosis or the so-called ‘ button-hole slit.” .

ETIOLOGY.

All morbid changes in the blood, causing irritation to the free surface of the endocardium over which it passes; Bright's disease —from irritation produced by the urea in the circulation; and acute articular rheumatism (sometimes). Whenever it occurs, it is due to some changes in the salts of the blood, or to a distinct poison, acting as an irritant to the valvular surface of the endocardium. The primary seat of the inflammation is in the serous structure, and the changes in the fibrous framework are secondary. It may occur in diphtheria, septicæmia, pyæmia, and fevers of all kinds. Secondary syphilis, which causes catarrh of the uriniferous tubules and amyloid kidney, may thus tend to create it.

SYMPTOMS.

Rational.

Pain—in the joints (if rheumatism be present).

Palpitation—over the precordial space (if the muscular tissue of the heart is involved).

Pulse—is sharp, quick, and irregular; but feeble and compressible when myocarditis occurs.

Temperature—rarely over 103°.

Dyspnœa—slight; the respiration is usually somewhat accelerated.

Physical.

Inspection—Increased area of heart's impulse. Irregular heart's action—indistinct later on in the disease.

Palpation—Cardiac impulse is increased at first, and afterward decreased.

Percussion.—The precordial dullness is normal (until the cavities are distended with blood from feebleness of the heart); then the dullness is increased.

Auscultation.—Systolic, ventricular valvular murmurs are heard. If it is produced *at the mitral or tricuspid orifice*, it is due to the tumefied condition of their edges and shortening of

the chordæ tendineæ, or to adhesions. If it occur at the *aortic orifice*, it is due to a simple roughening of the endocardium covering the aortic valves, and a soft, blowing, systolic murmur is thus produced.

DIFFERENTIAL DIAGNOSIS.

First, determine if it is an old or recent murmur. If it should happen to be an old murmur, there will be *compensatory hypertrophy* of the heart. If it be due to acute rheumatism, there will be an endocardial murmur, which will come on while the case is under your care. Should there be a murmur at your first visit, which is systolic, soft, and blowing in character, and *no hypertrophy*, it is produced probably by an acute endocardial inflammation: but if the murmur is rough in character, diastolic, and associated with *cardiac hypertrophy*, there is no evidence of acute endocarditis.

For the differential diagnosis from pericarditis (see acute pericarditis).

PROGNOSIS.

Is good, so far as imminent danger to life is concerned, but bad as regards complete recovery. Is rarely fatal, if occurring in Bright's disease, or rheumatism (still there will always be permanent valvular lesions). Is fatal, if it occur in pyæmia, septicæmia, and diphtheria (the danger being perforation of the valves or embolism). The organs which are most likely to be affected from an embolus, during this disease, are the spleen, kidneys, and the brain. Embolism of the spleen is characterized by pain or swelling over the spleen; pain in the loins and albuminuria mark a similar condition of the kidney; sudden hemiplegia occurs from a similar brain complication. If ulcerations occur at the free border or base of the valves, they may cause a rupture or tearing of the valves; following this there is a tendency to weakness and typhoid symptoms, extreme dyspnœa, cyanosis, and a sudden harsh regurgitant murmur, with the first or second sound of the heart. The disease may prove fatal in a few hours, or at most from three to four days, if such a condition be developed; the duration of life depending upon the size of the abnormal opening.

TREATMENT.

If it be due to rheumatism, rheumatic treatment must be resorted to. If from pyæmia, septicæmia, or diphtheria, *quinine* and *iron* must be freely administered. If from fevers, the temperature must be held in check. If from Bright's disease, the urea must be eliminated. *Opium* should be given *moderately* in order *to secure rest. The patient must always be kept perfectly quiet;* the temperature of the room should never be below 70°; the chest should be covered with flannel, especially while examination of the heart is being made. The patient must be fed with the most concentrated nutrition. *Digitalis* should be administered if the heart become feeble. Iodide of potassium has been recommended to absorb the fibrinous exudation.

The most efficacious agents in the treatment of this disease will be found in *rest, opium, iron,* the most *nutritious diet,* and the occasional use of *stimulants.*

ULCERATIVE ENDOCARDITIS.

MORBID ANATOMY.

In this form of endocarditis the new cell-growth is so rapid that the connective-tissue is not formed and pus results; under these circumstances the endocardium either ulcerates, or the entire valve may be perforated.

ETIOLOGY.

It occurs in pyæmia, septicæmia, puerperal fever, and in diseases characterized by great vital depression.

SYMPTOMS.

Rational.

Chills—previous to the attack.
Pulse—90 to 150 (at the time of the attack).
Temperature—105° to 107°.
Dyspnœa—extreme and sudden, when perforation takes place.
Posture—sitting.

Jaundice—(possible).
Spleen—enlarged.
Urine—scanty, highly colored, and albuminous.
Typhoid symptoms before death.

PHYSICAL.

Auscultation.—A hard regurgitant murmur is heard with the first or second sound, coming on suddenly.

PROGNOSIS.

Usually fatal. Death may result in a few hours, or three to four days at most.

CHRONIC ENDOCARDITIS.

DEFINITION.

Is a parenchymatous inflammation, marked by thickening and induration of the endocardium (being of an acute origin); is principally found in the endocardial covering of the valves, and that portion lining the apex of the left ventricle. It occurs principally in persons of a rheumatic history.

MORBID ANATOMY.

THE THICKENING — is the immediate result of the increase of connective-tissue. It is most marked at the base of the valves and along the line of contact. It may be very slight, or the valves may be so roughened, thickened, and hardened, as to impair or entirely destroy their functional activity.

THE RETRACTION—is the result of the changes in the new connective-tissue formations, wherein it becomes of a fibroid character, causing rigidity, diminution in depth, and puckering of the valves. Their edges have a cartilaginous feel. The changes are most marked in the mitral valves, being chiefly detected at the base of the valves, and around the valvular orifices; the edges of the valves are sometimes drawn down and become fastened to the walls of the ventricles by a short tendinous cord, causing extreme regurgitation.

THE ADHESIONS—occur with the retractions; and commence at the edges of the valves (as above), and continue till all traces of the valves may be lost, producing the button-hole slit. This is usually confined to the mitral and aortic orifices.

THE DEGENERATION OF THE VALVES.—After the cell- and new connective-tissue formation have gone on, and reached a certain point, fatty, granular, or calcareous degeneration takes place, forming under the endocardium patches of fatty, granular, or calcareous substance. These result occasionally in ulceration, and their removal is usually followed by extensive destruction of tissue, rupture of the valves, and regurgitation. Calcareous degeneration is mostly in the aortic orifice, as the changes occur at a late period of life.

These growths are not superficial, but an increase in the tissue underneath the endocardium, being sometimes half an inch or more in length. They are termed vegetations, often cause sudden and fatal regurgitation. When separated from the valve, they give rise to emboli, and development of infarction, and death of some portion of the various vital organs. These vegetations are of a *fibroid-tissue* growth extending through the entire thickness of the cardiac walls, causing shortening, thickening, and contraction of the chordæ tendineæ and columnæ carneæ. They are due to inflammatory changes.

SYMPTOMS.

The manifestations of this condition are in the heart's sounds, indicating valvular changes.

PROGNOSIS.

Depends upon the seat, and extent of the valvular lesion.

TREATMENT.

Remove the cause. Remove your patient from all excitement and alcoholic stimulants. Protect the body from sudden exposure to temperature (see valvular lesions for further treatment).

VALVULAR LESIONS.

These lesions are of *two kinds.*

First.—VALVULAR THICKENINGS, with slight retraction and adhesions, and atheromatous and calcareous degenerations; which give rise to murmurs, and cause *obstruction* to the current of the blood.

Second.—EXTENSIVE VALVULAR RETRACTION, perforation, partial detachment of the valves, rupture of the chordæ tendineæ, and calcareous plates, causing the valves not to close, and thus allowing of a return of the blood to the cavity, called ' *insufficiency.*" These alterations may co-exist, one being more extensive than the other.

AORTIC OBSTRUCTION.

DEFINITION.

An abnormal condition of the aortic valve, obstructing the escape of blood from the left ventricle into the aorta. It is always accompanied by hypertrophy of the left ventricular wall.

MORBID ANATOMY.

Changes take place in the aortic valves during acute and chronic endocarditis, as well as atheromatous degeneration of these valves; causing them to become thick and rigid, and to protrude into the current, thus producing a murmur with the *first sound* of the heart. There may be also vegetations on the surface of the thickened endocardium on the valves, obstructing the outgoing current, or a development of calcareous plates underneath the valves, also producing obstruction (stenosis). All of these conditions involve the left ventricle, producing gradual hypertrophy of its walls (unattended by dilatation); and, after a time, insufficiency of the mitral valves follows (due to extension of the endocardial inflammation).

ETIOLOGY.

It occurs in middle or advanced life, from acute or chronic endocarditis; or, from prolonged and severe muscular exertion, and atheromatous degeneration of the aorta.

SYMPTOMS.

Rational.

Pulse—small in volume, compressible, jerking, or intermittent.
Syncope—(on account of cerebral anæmia)—may occur.
Embolism of the brain (if the vegetations become detached and pass upward).
Œdema of the feet (from defective venous return)—may occur.

Physical.

Inspection.—The area of cardiac impulse is increased.
Palpation.—The force of the heart is increased, and heaving; and the heart-beat is more to the left than normal.
Percussion.—Increased area of dullness (to the left and downward)
Auscultation.—There is a murmur with the *first sound*, directly over the aortic valve, behind the sternum at the junction of the third rib. The murmur is heard with the greatest intensity at the second costo-sternal articulation of the right side, and along the carotids. It may replace the first sound or follow it immediately. The murmur is harsh in character.

DIFFERENTIAL DIAGNOSIS.

Rests upon the presence of a systolic aortic murmur; it may be mistaken for mitral, tricuspid, aortic regurgitant, and anæmic bruit.

Aortic obstructive.

Is a systolic murmur, heard at the *apex*, and the anterior portion of the chest. The maximum of intensity is heard at the second costo-sternal articulation of the right side, and along the carotids.

Mitral regurgitant.

Is a systolic murmur heard at the base of the heart, with less intensity than at the apex. The maximum of intensity is heard at the apex, and carried round to the left.

Aortic obstructive.

Maximum of intensity (see above).

Tricuspid regurgitant.

The maximum of intensity is heard at the apex, and is rarely audible above the third rib with the sternum.

Aortic obstructive.

Hypertrophy of the heart exists. Increased force of apex-beat, and jerky pulse may be detected.

Anæmic bruit.

Is heard over the carotids and is a venous hum. There is a feeble apex-beat; the pulse is soft, full, and compressible.

PROGNOSIS.

If the rhythm of the heart is not destroyed, the chances as to duration of life are not bad; otherwise they are, as the patient may die of cerebral anæmia, from the cutting off of the blood-supply. If the murmurs have lasted one or two years, you may be sure there are vegetations.

TREATMENT.

See "Aortic Regurgitation."

AORTIC REGURGITATION.

MORBID ANATOMY.

This is one of the gravest forms of heart-disease. The semilunar valves may be shortened, or shrunken, by chronic endocarditis, preventing closure; or, there may be laceration, dilatation, or adherence, causing a free opening. The regurgitation causes over-distention of the left ventricle, producing dilatation of that cavity from relaxation of the muscular fibres; and hypertrophy of the left ventricle ensues, in order to resist, and overcome the obstruction to the circulation. Over-dilatation of the arteries also ensues from an abnormal quantity of blood being thrown into them. Endocarditis is developed; and degeneration of the arterial walls occurs, leading to the rupture of an artery, usually in the brain (in case of over-excitement or excessive exercise), and the symptoms

of cerebral apoplexy. Tissue-degeneration of the heart also takes place from diminished supply to the coronary vessels, through insufficiency of the aortic valves. Dilatation of the left ventricle recommences and progresses (extensively); the mitral valves are not strong enough to close the auriculo-ventricular orifice, and regurgitation takes place through that orifice, on account of an extension of the endocarditis, atheromatous change, chronic endocarditis, and shrinking or shortening of the chordæ tendineæ. Aortic regurgitation thus becomes modified, by failure of the mitral valves. There is a general dirturbance of the venous circulation, late in the disease, resulting in cyanosis and dropsy.

ETIOLOGY.

Acute or chronic endocarditis; bodily exertion; atheroma of the aorta.

SYMPTOMS.

Rational.

Palpitation of the heart—(which is not constant).
Hypertrophy—sometimes excessive, producing excessive heart's action, vertigo, headache, syncope, and spots before the eyes.
Pulse—quick, jerking, irregular, and intermittent.
Radial pulse—a little after the apex-beat, in point of time.
Dyspnœa—due to pulmonary congestion.
Anæmia—excessive (due to emptiness of the arteries).
Giddiness—(due to interference with the cerebral circulation).
Increase in the carotid pulsation.
Overloading of the veins (due to impaired return to the heart).
Cyanosis (when the lungs are excessively congested).
Dropsy (when the systemic circulation becomes impaired).
Death (from cerebral embolism, disease of liver and kidneys, apoplexy, failure of heart, general dropsy, etc).

Physical.

Inspection.—Increased area of the apex-beat, and carotid pulsation.
Palpation.—Heaving impulse about the 8th rib, and to the left of the left nipple.
Percussion.—Area of precordial dullness is to the left of, and below

the normal area, and is more oval than normal; as dilatation comes on, the dullness will extend upward, and the apex-beat may be perceived as high up as the axillary space.

Auscultation.—A murmur is heard with the *second-sound*, sometimes immediately following it; maximum intensity over the second intercostal space close to the right edge of the sternum over a large area, or down to the xyphoid cartilage, or, along the spine. It is heard, as a diastolic murmur, at the base of the heart; when combined with aortic obstruction, there is a double murmur heard over a large area, at the second intercostal space to the right, or left of the sternum.

DIFFERENTIAL DIAGNOSIS.

Rests on the diastolic murmur, and the presence of hypertrophy and dilatation. It can only be confounded with mitral stenosis (which has no hypertrophy, nor dilatation of the left ventricle). In pericarditis (confined to that portion above the aorta), a friction-sound resembling the murmur may be heard, but this occurs only during diastole, and without evidences of either hypertrophy or dilatation.

PROGNOSIS.

It depends on the age of the patient. It is exceedingly unfavorable when occurring in those of middle age, especially if they are engaged in active pursuits. The dangers are hypertrophy of the left ventricle, augmenting the distended force of the arterial walls, causing degeneration; degeneration of the hypertrophied ventricular walls, from impaired nutrition, on account of interference of the coronary circulation, causing excessive dilatation and weakening of the heart; and an inability of the heart to overcome the resistance, resulting in cyanosis, dropsy, and death. In old people, it gives rise to very little inconvenience.

CAUSES OF DEATH.

From hypertrophy and degeneration of the ventricular walls; from cerebral embolism; from regurgitation, accompanied by vegetations on the valves; from obstruction to the systemic circulation (producing sclerosis of the liver and kidneys); from pul-

monary œdema; cerebral apoplexy; or from sudden syncope (from obstruction to the return circulation).

TREATMENT.

Absolute rest is imperative. There must be avoidance of all physical and mental exercise, or intemperance in any form. The patient must have a nutritious diet, chiefly albuminous. Care must be taken not to disturb the heart's action. The patient must lead a quiet life. *Digitalis* should *never* be resorted to in this disease. *Iron* should be administered if there are symptoms of imperfect heart-power, and anæmia be present. For the relief of dyspnœa and dropsy, see "mitral regurgitation."

MITRAL OBSTRUCTION.

DEFINITION.

Is a condition of stenosis, or obstruction, of the auriculo-ventricular orifice of the left heart; and is due partly to constriction at the base of the mitral valves, and partly to adhesion of the valve-tips and chordæ tendineæ, following rheumatic endocarditis (it occurs principally in young children).

MORBID ANATOMY.

The new connective-tissue, by contracting, causes obstruction to the flow of the blood from the auricles to the ventricles, by producing incomplete opening of the auriculo-ventricular orifice. In stenosis, the edges of the valves become adherent, as may also the chordæ tendineæ, forming the "button-hole slit" (the normal opening should easily admit the ends of three fingers). Dilatation and hypertrophy of the left auricle are thus produced from its over-distention; also obstruction in the pulmonary veins, and dilatation of the pulmonary capillaries, causing congestion of the pulmonic circulation, or pulmonary congestion. The lungs may be found to be in a state of pigmentation ("brown induration"). Bronchorrhœa may also occur, due to hyperæmia of the bronchial mucous membrane; and pulmonary apoplexy may even occur from rupture of the pulmonary blood-vessels. This latter complication may be

caused by exercise, since the pulmonary hyperæmia is thus increased. Pulmonary œdema may be readily developed when this valvular disease is strongly marked, from walking against a strong head-wind; and may result in death.

ETIOLOGY.

Mitral obstruction may be the result of acute or chronic endocarditis, in young people; and of all the morbid changes in the blood; such as Bright's disease (from the irritation produced by urea in the circulation); acute articular rheumatism; sometimes of changes in the salts; or poisons, acting as an irritant to the valves (as in diphtheria, septicæmia, pyæmia, and all the fevers).

SYMPTOMS.

Rational.

Dyspnœa—(a result of the pulmonary congestion).
Cough—dry, hacking, unsatisfactory; resembles a nervous cough; is due to changes in the lungs (congestion), not to the heart.
Hæmoptysis—of dark color, usually slight in amount.
Expectoration — profuse, watery, and blood-stained (especially after exertion)—this denotes pulmonary congestion.
Pulse—regular (if the ventricle be not affected, and only a slight stenosis exist); but may be feeble (if stenosis be extensive).

Physical.

Inspection.—Feeble cardiac impulse.
Palpation.—*A distinct purring thrill* precedes the apex-beat.
Percussion.—Area of dullness increased upward, and to the left.
Auscultation.—A loud *blubbering murmur* is heard, just *before the first sound;* and is synchronous with the contraction of the auricle. The maximum of intensity is heard a little *above the apex-beat;* is louder and longer than any other murmur.

DIFFERENTIAL DIAGNOSIS.

Depends upon the *loud blubbering murmur*, and the *purring thrill.* It is a *presystolic* murmur. It may be confounded with *mitral* and *aortic regurgitation.*

PROGNOSIS.

Is bad, especially if excessive dyspnœa be present.

TREATMENT.

Same as in mitral regurgitation (see page 126).

MITRAL REGURGITATION.

DEFINITION.

Is caused by thickening, induration, and shortening of the mitral valve; due to changes produced by acute or chronic endocarditis. It is the most common valvular lesion.

MORBID ANATOMY.

The regurgitation is often caused by calcareous matter imbedded in the valves. The chordæ tendineæ become thick and short, and the papillary muscles (which are the terminations of the chordæ tendineæ) become diminished in bulk. The valves may be torn, the chordæ tendineæ ruptured, and the regurgitant current then causes them to flap; or the valves may become adherent to the ventricular walls through shortening of the chordæ tendineæ. The following changes take place in consequence:

1st.—*Dilatation of the left auricle* (as a result of excessive blood-pressure).

2d.—*Compensatory hypertrophy of the left auricle* (to increase its power, and thus to force blood through the narrowed orifice).

3d.—*Disturbed pulmonary circulation*, producing congestion or brown induration (when the previous hypertrophy proves insufficient).

4th.—*Hypertrophy of the right ventricle* (to overcome pulmonary obstruction).

5th.—*Dilatation of the right ventricle* (as the result of too great pressure).

6th.—*Tricuspid regurgitation* (due to an endocarditis excited, or to excessive dilatation of the ventricle).

7th.—*Dilatation of the right auricle* (from the pressure of the regurgitated blood).

8th.—*Compensatory hypertrophy of the right auricle* (to overcome the previous dilatation).

9th.—*Interference to the return circulation* of the venous blood from the superior and inferior venæ cavæ.

In superior vena cava.

Producing, headache; vertigo; jugular pulsation; prominent jugulars; cyanosis; œdema; apoplexy; etc.

In inferior vena cava.

Producing, nutmeg-liver; obstruction to the portal circulation; hemorrhoids; menstrual disturbance; gastric catarrh; epigastric pulsation (which is hepatic, from regurgitation into its vessels); intestinal catarrh; ascites; jaundice; enlarged spleen; passive hyperæmia of the kidney, or desquamative nephritis (catarrhal); and general anasarca.

In order to overcome these changes and keep up the heart's power, hypertrophy and dilatation of the left ventricle finally take place.

ETIOLOGY.

Primary cause.

Acute endocarditis.

It may be secondary to

some lesion of the aortic valves, either from extension of the endocardial inflammation or to a valvulitis excited by the regurgitant current from the aorta. Enlargement of the left auriculo-ventricular orifice, which accompanies excessive dilatation of the left ventricle, may produce it.

SYMPTOMS.

Rational.

Are chiefly due to the regurgitation at the tricuspid orifice—and are therefore developed late in the disease.

Cough—with watery expectoration which is sometimes stained with dark or blackish blood (a symptom of pulmonary dropsy).

Enlarged liver (due to interference with its return of blood).
Pain and weight in the right hypochondrium (from hyperæmia).
Jaundiced hue to the surface.
Cyanosis—(a symptom of impaired venous return).
Cerebral hyperæmia.
Headache—due to hyperæmia of brain.
Vertigo—due to hyperæmia of brain.
Stupor—due to hyperæmia of brain.
Delirium (temporary).
Gastric and intestinal catarrh—due to hyperæmia.
Hemorrhoids—due to hyperæmia.
Amenorrhœa—due to hyperæmia.
Urine—scanty, high-colored, and casts.
Rapid ascites—produced by a sero-albuminous exudation through the walls of the vessels; commencing in the lower part of the abdomen and extending upward.
General anasarca (beginning at the ankles and extending upward).
Irregular radial pulse—being at first exceedingly feeble, compressible, and tremulous on excitement.

PHYSICAL.

Inspection and Palpation.—Increased area of cardiac impulse, which may be forcible, or diffused; depending upon the amount of hypertrophy and dilatation. Apex-beat is more to the left than normal.

Percussion.—*Precordial dullness increased,* extending to the left beyond the nipple and downward; showing more or less hypertrophy and dilatation. *Area of superficial dullness is increased laterally* and *downward.*

Auscultation.—A murmur takes the place of, or follows, the *first sound,* and is synchronous with the systole of the ventricle, and due to regurgitation from the left ventricle to the auricle.

Diagnostic points.

Its greatest intensity is at the apex-beat. Area of diffusion is *to the left,* and it is heard with nearly the same intensity behind, between the fifth and eighth dorsal vertebræ, close to the left side of the spinal column. The *second*

sound is abnormally intense over the pulmonic valves a little above the third rib.

CAUSES OF DEATH.

Œdema of the lungs, on account of the feeble and irregular heart action; hemorrhagic infarction (from pneumonia), the inflammation causing death; general anasarca; or Bright's disease.

DIFFERENTIAL DIAGNOSIS.

From aortic regurgitation and obstruction, and tricuspid regurgitation; but the murmur, area of diffusion, and character of the pulse, are sufficient to enable you to make a satisfactory diagnosis.

In mitral regurgitation—the pulse is feeble and easily accelerated.

In aortic regurgitation—the pulse is hard and jerky.

In mitral regurgitation—the murmur occurs with the *first sound*, is heard at the apex and *carried to the left* and *backward* between the fifth and eighth dorsal vertebræ.

Aortic obstruction—occurs with the first sound and is *conveyed along the carotids.*

Tricuspid regurgitation—occurs with the first sound and is heard over the right ventricle and to the right of the area of the heart.

PROGNOSIS.

As far as the duration of life is concerned, the prognosis is good, on account of compensating hypertrophy and the stationary character of the exciting cause. If œdema, cyanosis, extreme dyspnœa, etc., be present, the prognosis is bad.

TREATMENT.

The patient must lead a quiet life, free from all excitement; and have absolute self-control. A change of occupation may become necessary, if there be a predisposition to pulmonary hypertrophy or bronchial catarrh. Forbid the use of the voice, as in singing, etc., as there is a constant pulmonary hyperæmia, which such acts tend to increase. If excessive pulmonary œdema occur, *venesection* or *hydragogue cathartics* should be resorted

to. Give *Inf. Digitalis* ʒ ss. every two hours for twenty-four hours; it relieves the hyperæmia and favors systemic circulation; it regulates and increases the systolic action, by its action on the muscles of the heart; it strengthens the right heart and enables the left ventricle to resist the increasing dilatation. *It should not be continuously employed,* but only for twenty-four or forty-eight hours, if good results be obtained. If it be judiciously used, it may cause disappearance of the pulmonary œdema, cyanosis, anasarca, and scanty urine.

Iron may be given with the food in full doses, but only in cases of anæmia (Vallet's Mass gr. x.–xx t. i. d.)

VALVULAR DISEASES OF THE RIGHT HEART.

These are all secondary to those of the left heart, and are very rare, as endocarditis seldom occurs on that side. When endocarditis does occur on the right side, it is usually confined to the tricuspid valve; and is due to obstruction in the pulmonic circulation from disease of the mitral valve, which induces an hypertrophy of the right ventricle and subsequent valvular lesions.

PULMONIC VALVES.

PULMONIC OBSTRUCTION OR STENOSIS.

Is usually caused by mediastinal tumors pressing on the pulmonary artery, diminishing its calibre and obstructing the current. The murmur occurs with the first sound, and the maximum of intensity is over the seat of the valve. The murmur is superficial and distinct, and the area of diffusion is toward the left shoulder.

PULMONIC REGURGITATION

Is doubted by many eminent physicians, if this condition exist to such an extent as to constitute a pathological lesion. The

murmur occurs with the second sound, over the pulmonic valves; and the area of diffusion is carried down to the xiphoid cartilage. There is no jerking or pulsation as in aortic regurgitation.

PROGNOSIS.

Is theoretically bad. If it occur as a primary disease, it causes dilatation of the right ventricle, tricuspid regurgitation, and disturbance of the systemic circulation.

TRICUSPID REGURGITATION.

DEFINITION.

Is usually secondary to mitral stenosis or regurgitation. It is preceded, as a rule, by hypertrophy of the right ventricle, since pulmonic obstruction has to be overcome.

MORBID ANATOMY.

There is thickening and shrinking of the valves; shortening of the chordæ tendineæ; and more or less thickening and induration at the base of the valves, thus diminishing their size. There is first dilatation and, secondly, a compensatory hypertrophy of the right auricle; there is disturbance in the circulation of the venæ cavæ, as shown under the description of morbid changes due to mitral regurgitation (page 123); last of all, the left ventricle becomes involved, on account of the increased work of the left heart from the *systemic* obstruction.

ETIOLOGY.

Its spontaneous occurrence is doubtful; pulmonary emphysema causing interference with the pulmonary circulation and subsequent endocardial inflammation, from abnormal stress thrown on the valves at the auriculo-ventricular orifice, may produce it; and also interference with the pulmonary circulation from any cause, as mitral lesions, mediastinal tumors pressing upon the pulmonary artery, etc.

SYMPTOMS.

Rational.*

When the venous return is interfered with, there may be

Enlarged liver—(due to venous engorgement)—nutmeg liver.
Dinginess on the surface of the body.
Urine—scanty.
Bright's disease (from passive hyperæmia of the kidney).
Headache (from passive hyperæmia of the brain).
Vertigo (from passive hyperæmia of the brain).
Dementia (from passive hyperæmia of the brain).
Irregular heart's action.
Cardiac palpitation.
Dyspnœa (from passive hyperæmia of the lung).
Face—blue and turgid; with stupor, coma, and cerebral compression, *if the patient be placed in a horizontal position.*

Physical.

Inspection.—Increase in the visible cardiac impulse from the apex-beat to the xiphoid cartilage and to the second intercostal space. *Pulsation in jugulars. Distended jugulars.*

Palpation.—Indistinct apex-beat, unless hypertrophy of the left ventricle exist. *Epigastric pulsation.*

Percussion.—Area of dullness is increased to the right of the sternum and to the second intercostal space.

Auscultation.—There is a *blowing murmur* taking the place of the first sound. It is *superficial* and *rarely heard above the third rib.* The maximum of intensity is *between the fourth and and sixth ribs* at the *left border* of the sternum. The *second sound* is *increased in intensity*, and heard most distinctly over the pulmonic valve.

DIFFERENTIAL DIAGNOSIS.

From mitral regurgitation; aortic obstruction; and pulmonic obstruction.

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* For the explanation of these symptoms see page 124 of this volume.

PROGNOSIS

Is bad (see page 124 of this volume, where dangers, due to venous engorgement, are given in some detail).

TREATMENT.

The patient must lead a quiet life, and reside in a warm climate; where a free action of the skin can be maintained. When it occurs in connection with emphysema, *digitalis* is not well borne, as it increases the jugular pulsation and distends the veins of the head and neck, but, should it occur in connection with mitral regurgitation, *digitalis* is then of great service. *Iron* must be administered, if anæmia exist (as mentioned under the treatment of mitral regurgitation). *Drastic cathartics* temporarily relieve the venous engorgement and cerebral oppression, but, should anasarca be developed, incision or needle pricking is the only means of relief.

CARDIAC MURMURS.

RHYTHM OF MURMUR.

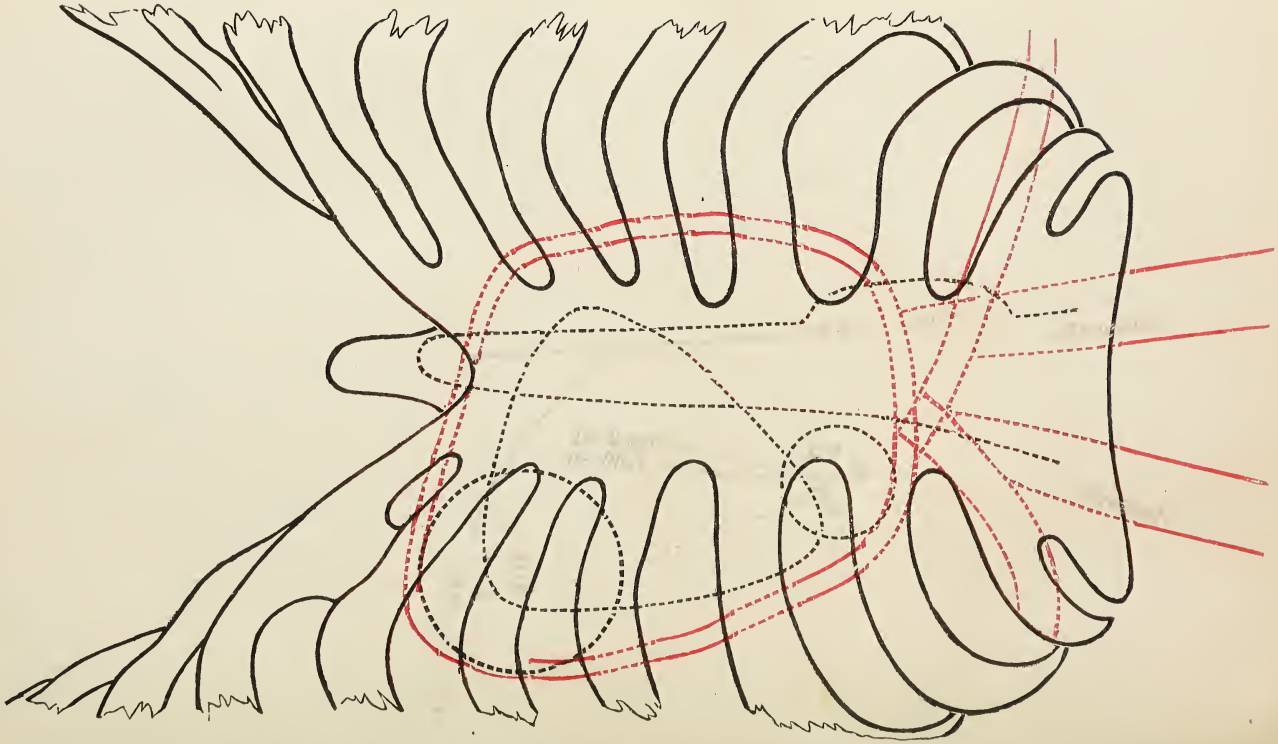
Is the relation of the murmur to the *sounds of the heart.*

HEART-SOUNDS.

First sound.—Is distinguished by the striking of the apex of the heart against the chest-wall, and is synchronous with the radial pulse. It is longer in point of duration than the second sound, since it is due to the continued effect of four causes, as follows: (1) The blow of the apex upon the chest-wall; (2) the friction of the muscular fibres of the ventricles; (3) the rush of blood in the ventricles; (4) the closure of the auriculo-ventricular walls. Should there be a murmur it will bear the same relation to the apex-beat and radial pulse as the first sound does.

Second sound.—Is distinguished by a short, sharp sound which is produced by closure of the aortic and pulmonic valves.

All murmurs either precede, take the place of, or follow immediately one of the sounds of the heart.



PRESYSTOLIC MURMURS.

Mitral obstructive.—Is heard with greatest intensity over a circumscribed space at the apex. It is characterized by a *purring* sound, and is the loudest and longest of all the murmurs.

Tricuspid obstructive.—Is heard with greatest intensity along the margin of the fifth and sixth costal cartilages, on the left border of the sternum. Its area of diffusion is over the right ventricle and rarely above the third rib.

SYSTOLIC MURMURS.

Mitral regurgitant.—Is heard with greatest intensity at the apex. Its area of diffusion is *carried to the left*, and is often heard between the fifth and eighth dorsal vertebræ or at the angle of the scapula.

Tricuspid regurgitant.—Is heard with greatest intensity between the fifth and sixth ribs along the sternum. Its area of diffusion is over the right ventricle and *rarely* above the third rib. It is a *loud blowing murmur.*

Aortic obstructive.—Is heard with greatest intensity at the second costo-sternal articulation of the right side. Its area of diffusion is *upward along the carotids.* Inaudible at the apex.

Pulmonic obstructive.—Is heard with greatest intensity over the seat of the valves at the junction of the third rib and sternum of the left side. Its area of diffusion is carried *toward the left shoulder.*

DIASTOLIC MURMURS.

Aortic regurgitant. — Is heard with greatest intensity at the third costo-sternal articulation on the right side. Its area of diffusion is carried *down the sternum* to the xiphoid appendix, and it *may be* heard at the apex.

Pulmonic regurgitant. — Is heard with greatest intensity over the seat of the valve, at the junction of the third rib and the sternum of the left side, and is carried down to the apex.

In addition to the above-mentioned murmurs there may also be

Anæmic murmurs—heard with greatest intensity *in the carotids.*

Ventral murmurs—are produced within the left ventricle from roughening of the chordæ tendineæ, or the ventricular sur-

face of the mitral valves, or by an abnormal direction to the current of blood as it passes through the ventricle. These are very rare.

CARDIAC HYPERTROPHY.

DEFINITION.

Is the result of overwork, and consists of a thickening of the walls of the heart by an increase in the muscular tissue, involving both the auricles and the ventricles, or either one alone.

VARIETIES.

Simple—where there is thickening of the walls, but *no increase in the capacity of the cavities;* it is confined to the left ventricle (occurs in Bright's disease and alcoholism).

Eccentric—where there is thickening of the walls, *and increase in the capacity of the cavities* (occurs with valvular lesions).

Concentric—where there is thickening of the walls, but *diminution in the capacity of the cavities;* this is of very rare occurence.

MORBID ANATOMY.

In the eccentric form.—There is an increase in the size of the papillary muscles, and thickening of the septum. A change takes place in the shape of the organ, which corresponds with the seat of the hypertrophy. If the hypertrophy be confined to the left ventricle, the heart will be of a pyriform shape: if to the right ventricle, there is an increase in the horizontal measurement, making it oval and elongated. The walls of the heart are stiffened, and do not collapse when opened after death; and are redder than normal. Hypertrophy is simply a hyperplasia. There may be an increase in the size of the muscular fibres, but the hypertrophy chiefly consists in an increase in their number. The heart may weigh 40 ounces more than normal.

ETIOLOGY.

It seldom exists without valvular lesions, arterial changes, or capillary obstruction. Anything which tends to create dilatation of the heart cavities or mechanical obstruction to the circulation of blood may produce it.

SYMPTOMS.

Rational.

In Moderate Cases.

Fullness of the arteries.
Emptiness of the veins.
Pulse—full and strong.
Face—easily flushed; and eyes brilliant.
Carotid pulsation.

In Severe Cases.

A sense of fullness in the chest, from pressure of the enlarged heart.
Uneasiness—in the epigastric region—from pressure.
Impaired digestion—from pressure.
Dyspnœa—from pressure on the lung.
Cardiac palpitation—under excitement.
Headache—when under excitement.
Vertigo—when under excitement.
Tinnitus aurium—when under excitement.
Atheroma—from extension of the endocarditis.
Endarteritis—from extension of the endocarditis (causing changes in the coats of the vessels).

Physical.

Inspection.—Increased and distinct area of impulse, and visible motion of the chest on cardiac pulsation. *In children*—bulging of the precordial space, and displaced apex-beat.

Palpation.—Increased area of apex-beat and the detection of a heaving, lifting character over the whole precordial space; which determines hypertrophy of the left ventricle. If hypertrophy of the right ventricle exist, there will be a strong *epigastric pulsation.* If hypertrophy of the left ventricle exist, the apex-beat will be to the left (three inches below and two or three inches to the left of the nipple). *In eccentric hypertrophy.*—If the right ventricle be affected, the apex-beat is to the right and downward. If the left ventricle be affected, the apex-beat is to the left and downward.

Percussion.—Dullness is increased to the right, or left, or downward; and when upward the *auricles are hypertrophied* and

dilated, but it is rare. If hypertrophy of the right ventricle exist, there is dullness to the right of the sternum. If hypertrophy of the left ventricle exist, there is dullness to the left beyond the nipple. If *eccentric*—the dullness is increased in both directions.

Auscultation.—The *first sound* (if no murmur be detected) will be dull, muffled, and prolonged, with increased intensity. The *second sound* is increased in intensity in both hypertrophy of the left and right ventricle. If there be hypertrophy of the left ventricle, it is heard with greatest intensity over the aortic orifice; if there be hypertrophy of the right ventricle, it is heard with greatest intensity over the pulmonic orifice. There is *absence of the respiratory murmur* over the precordial space. In emphysema, there may not be much increase of force in the apex-beat; the heart's sounds will be diminished; and a venous pulsation in the neck (due to hypertrophy and dilatation of the right ventricle with tricuspid regurgitation) may be perceived.

DIFFERENTIAL DIAGNOSIS.

In *eccentric hypertrophy of the left ventricle.*—The pulse will be full and strong; there will be carotid pulsation, the countenance flushed; the eyes prominent and brilliant; the apex-beat forcible, over an unnatural area, and carried to the left; the cardiac dullness increased to the left and downward; and there will be increased intensity of heart's sounds, especially of the second sound.

In eccentric hypertrophy of the right ventricle, there will be forcible heart's action noticed along the sternum and left lobe of the liver; the apex-beat is carried to the right and downward; the cardiac impulse is nearer the median line; there is somewhat of an *epigastric impulse,* increased area of dullness to the right; and the pulmonic sounds are increased. The first sound is more intense than normal and nearer the median line.

In total eccentric hypertrophy.—Everything is the same as in hypertrophy of the left ventricle, except that the dullness is increased in all directions, and the heart's sounds are more intense than normal.

The condition of hypertrophy is to be further differentiated from cardiac dilatation; thoracic aneurism; mediastinal tumors; consolidation of the lung-tissue; and pleuritic effusions.

PROGNOSIS.

Is more favorable than in any other cardiac affection; simple hypertrophy (unless from aortic stenosis) may exist for years; slight hypertrophy is very common; if degeneration of the hypertrophied walls exist, the prognosis is very unfavorable. When hypertrophy of the right ventricle is accompanied by pulmonary obstruction, the prognosis is bad.

TREATMENT.

Alcoholic stimulants, immoderate eating, active and prolonged physical and mental exercise, must be strenuously avoided. All obstructions to the abdominal circulation must be removed. The bowels should be made to move freely, by regulating the diet so as to prevent constipation and straining. If cerebral symptoms occur, they should be overcome by *aconite in full doses. Fleming's tincture of the root*, gtt. ij.–iij. may be given every three or four hours, but should the dilatation exceed the hypertrophy, do not give it. *Digitalis* should only be administered when the heart's action is enfeebled.

CARDIAC DILATATION.

DEFINITION.

A condition which is characterized by an increase in the capacity of its cavities, and a diminution in its contractile power.

VARIETIES.

Simple—Where there is an increased capacity of the cavities and *no marked change* in the walls; it occurs in convalescence from typhoid fever, or any disease where there has been great impairment of nutrition.

Hypertrophous—Where there is an increased capacity of the cavity; increased thickness of the heart-walls; and *diminished contractile power.*

Atrophic—Where there is a marked increase in the capacity of the cavities; the cardiac walls are thinner than normal; the *ventricular walls diminished to two or three lines*, and the auricles are almost transparent.

MORBID ANATOMY.

This condition may occur in one or all the cavities. The heart will be altered in shape, and this will depend upon where the seat of dilatation is situated. It occurs most frequently in the auricles; next in the right ventricle; and lastly in the left ventricle. If the walls of the left ventricle be very much thinned, they collapse when the ventricle is cut. A heart that is distended with blood and relaxed by putrefaction may be easily mistaken for a dilated heart; but, in such instances, the heart is extremely soft and saturated with the coloring matter of the blood, and there will be evidences of decomposition in other parts of the body.

ETIOLOGY.

First cause —From *internal pressure during cardiac diastole;* from weakness, as the result of prolonged disease; from fatty degeneration; from abnormal pressure, producing permanent dilatation of its cavities; from degeneration, and an absence of compensatory hypertrophy.

Second Cause.—From loss of tone when the muscular tissue of *the heart is the seat of primary fatty degeneration;* as occurs when there is first dilatation of the cavities with excessive blood-pressure, and after myocarditis.

Third Cause.—From degeneration of muscular substance of the heart, which is the seat of *eccentric hypertrophy* (occurring after valvular diseases).

SYMPTOMS.

RATIONAL.

In simple dilatation.

The cavities are increased, and labored heart's action exists.

No increase in the heart's force.

Feeble radial pulse.

Rhythm not disturbed.

In atrophic dilatation.

The cavities are dilated and thinned.

Labored heart-action and feeble power.

Marked feebleness of the radial pulse.

Heart staggers—from increased amount of blood.

Arteries—imperfectly filled.

Veins—distended and engorged.

Disturbed rhythm.

Radial pulse—is weak and intermittent.

Common symptoms.

Cardiac palpitation—severe, and distressing syncope.

Constant and painful sense of pulsation of the heart.

Dyspnœa—on slight exertion (which is constant in bad forms of this condition).

Countenance—pale, languid, and anxious.

Lips—livid, even when the patient is quiet.

On excitement.

Face and neck are livid.

Pulse—irregular and intermittent.

Loss of mental powers.

Dyspeptic symptoms.

Fullness in the epigastrium.

Urine—scanty and albuminous.

Œdema—causing the patient to sit up with the head forward, and resting on some support.

Cyanosis—(when extensively developed).

Yellow tinge of the surface—from disturbance of the liver.

Delirium—(a late symptom).

Death—from syncope, anasarca, or pulmonary œdema

PHYSICAL.

Inspection.—Indistinct and *increased area* of impulse, (especially in fat or œdematous people). *Undulating movement* over the precordial space (in thin subjects). No prominence of the precordial region. Irregular and *distended jugulars* (if the right heart be distended).

Palpation.—Feeble cardiac impulse; absence of the lifting, forcible impulse, as in hypertrophy. *Apex-beat is dif-*

fused, undulating, wanting in power, and resembling a feeble step. *Purring thrill* with the apex-beat like mitral stenosis, if mitral regurgitation co-exist.

Percussion.—Increased dullness laterally (one inch to the right, if the right side be involved; or one inch to the left, if the left side be involved, often extending into the axillary space). The precordial dullness is *oval in shape.* The area of *superficial dullness is not increased*, as in hypertrophy. If the auricles be dilated, an upward increase in the area of dullness exists. If the *jugular veins* be permanently knotted, and dilated, there is dilatation of the right auricle.

Auscultation.—The first and second sounds are short, feeble, and *nearly of equal duration;* and the second sound is often inaudible at the apex-beat. Asystolism is often present after exercise.

DIFFERENTIAL DIAGNOSIS.

Rests upon the feeble action; undulating impulse; indistinct apex-beat; lateral increase in the area of percussion dullness; short, abrupt, feeble heart's sound; feeble, irregular, and intermittent pulse; and the general symptoms of systemic and pulmonic obstruction.

The differentiation must be made between cardiac hypertrophy and cardiac dilatation; pericarditis with effusion, and cardiac dilatation; enlargement from dilatation or hypertrophy, in connection with thoracic aneurism or mediastinal tumors.

In thoracic aneurism or mediastinal tumors—there is the direction of the increased area of dullness to assist, since it *is always upward*, and to the right or left.

In cardiac enlargement there is increased area of dullness, *laterally*, and *downward*. *Consolidation of lung-tissue* may give rise to some of the signs of cardiac enlargement.

PROGNOSIS.

Is bad, and the danger increased in proportion to the excess of the capacity of the cavities over the thickness of their walls. There is danger of death from dyspnœa and syncope.

TREATMENT.

This is an incurable disease. In order to prevent flaccidity of the walls of the heart, nutrition must be kept up to its highest point by a milk diet and stimulants; and, if there be anæmia, administer *iron*. Plenty of fresh air, the best possible hygienic surroundings and stimulating baths, to increase the capillary circulation, should be afforded. All irregular and violent exercise must be forbidden; flannels must be worn next to the skin; arrest all exhausting discharges; and, as the various complications in the liver and other abdominal organs arise, they must be treated separately. If there be loss of appetite, etc., give vegetable tonics and mineral acids. *Digitalis* is very serviceable in this disease *in full doses*, but if it cannot control the heart's action, then *belladonna* and *opium* should be given *in combination with digitalis*.

In the paroxysms of dyspnœa, *hydrocyanic acid*, *cannabis indica*, or *ether* may be given; and dry cupping along the spine. The drugs and treatment upon which most reliance can be placed are *digitalis* and *iron*, and a *good wholesome nutritious diet*.

MYOCARDITIS.

DEFINITION.

Is an inflammation of the *muscular structure* of the heart; associated with degeneration and softening, which may be of two varieties:

1. *General* or *diffused*—which is rare.
2. *Local* or *circumscribed*—which is met with in connection with pericarditis and endocarditis; usually involving only the internal or external surface of the heart.

MORBID ANATOMY.

The left ventricle is most frequently affected; the muscles changing from dark-red to gray, and finally to dark-green. It terminates either in a connective-tissue formation, or in abscess. The connective-tissue causes the power of resistance of the ventricular wall to be diminished. This connective-tissue during diastole is gradually and slowly stretched; and thus, finally,

aneurism of the heart results. When the inflammatory process becomes degenerative, abscesses form, and rupture of the heart may take place.

ETIOLOGY.

Predisposing Causes.

Rheumatism—terminating in connective-tissue formation.
Endocarditis.
Syphilis.
Temperature—which must be high and continuously so.
Extensive, and long continued, pericarditis.

Exciting Causes.

Embolism of the coronary arteries.
Pyæmia (terminating in abscess).
Septicæmia.
Typhus fever.
Typhoid fever.

SYMPTOMS.

Rational.

Cardiac palpitation, and pain in the precordial region.
Pulse—feeble, irregular, and intermittent.
Syncope—on exertion.
Failure of the heart's action.

Physical.

Palpation.—The area of the precordial dullness is increased *upward* and *toward the left shoulder*, as in hypertrophy, but there is no heaving impulse.

PROGNOSIS

Is rapidly fatal if abscess occur; more slowly so, if the new connective-tissue formation is prominently developed.

TREATMENT.

Absolute quiet should be enforced, and the nutrition carried to its highest point. All active and prolonged physical exertion must be prohibited.

FATTY DEGENERATION OF THE HEART.

DEFINITION.

True fatty degeneration of this organ is a substitution of fatty matter in place of the muscular tissue of the heart, to such an extent as to interfere with its normal action.

VARIETIES.

1st. There may be a *fatty degeneration* of the primitive muscular fibre (Quain's).

2d. There is a *fatty deposit* in the areolar or connective-tissue of the heart which is simply replaced by fat, the muscular fibres being unaffected.

MORBID ANATOMY.

"*In Quain's fatty degeneration*"—the degeneration is confined to the muscular fibre, the primitive fibre-bundles lose their nuclei, their striated appearance disappears, and they become granular. The sarcolemma becomes filled with granules, and the sarcous substance gives place to fat-granules and oil-globules. The muscular-tissue assumes a yellow or dirty-brown color, and breaks down very easily under pressure. The coronary arteries may be atheromatous, calcified, obliterated, or normal.

In the second form—there may simply be an increase of fat in the areolar tissue of the heart which does not interfere with the condition of the muscular fibre, except by its pressure; hence the heart is simply flabby, pale, or yellowish, and may be more bulky than usual.

ETIOLOGY.

It may be a true fatty metamorphosis, due to interference of the heart's nutrition; or, secondly, an adipose condition of the heart due to an excess of fat in the blood (although this is not, properly speaking, a degenerative lesion).

Predisposing Causes.

(1) *Malnutrition*, and interference with the *coronary arteries.*
(2) Excessive or perverted nutrition (causing a deposit of fat in the organ); or hereditary predisposition.

Exciting Causes.

Bright's disease.
Chronic alcoholismus.
Gout.
Phthisis.
Cancer.
Calcification of the coronary vessels.
Pericardial thickenings—causing external compression.
Poisoning by phosphorus, phosphoric acid, chloroform, etc.
General obesity.

SYMPTOMS.

Rational.

The same as those of defective heart-power.

Complete exhaustion after exercise.
Skin—pale, sallow, and livid.
Arcus senilis.
Enlarged liver.
Muscular flabbiness.
Respiration—feeble and irregular, often sighing in character.
Disturbed vision—from anæmia of the brain.
Irritable temper—from anæmia of the brain.
Dementia.
Dyspnœa—from pulmonary hyperæmia.
Giddiness—from anæmia or passive hyperæmia of the brain.
Vertigo—from anæmia or passive hyperæmia of the brain.
Syncope—from anæmia or passive hyperæmia of the brain.
Feeble Pulse.

Physical.

Inspection.—Feeble, sighing, or progressive respiration. Apex-beat is *indistinct* and often invisible.

Palpation.—*Tumbling, rolling motion* of the heart (if hypertrophied).

Percussion.—Increased area of dullness (which is both superficial and deep), if hypertrophied—otherwise normal limits.

Auscultation.—First sound is feeble or absent. *Second sound* is feeble but distinct.

DIFFERENTIAL DIAGNOSIS.

Between cardiac dilatation, and simple fatty heart.

PROGNOSIS.

There is always a fatal termination. This may occur from syncope, rupture of the heart, cerebral anæmia, or general dropsy.

TREATMENT.

Iron, oleum morrhuæ, good nutritious diet, fresh air, light exercise, and the avoidance of all stimulants is all that can be suggested for the treatment of this disease, as the great and only object, to be obtained, is to improve, or increase the tissue-making power of the blood.

CARDIAC THROMBOSIS.

MORBID ANATOMY.

Heart-clots may have existed for years, or only a short time previous to death; and vary in size from that of a pin-point to that of a walnut. They may be flat or round in shape. When small they are called vegetations, and when large, thrombi. They may be found in either of the cavities, or on the valves.

ETIOLOGY.

First—you may have obstruction to the flow of blood, which may be due to valvular lesions, dilatation, or feeble contractile power of the heart.

Secondly—you may have abnormal changes in the composition of the blood.

Thirdly—you may have endocarditis (causing a roughening of the endocardial membrane and subsequent formation of thrombi).

SYMPTOMS.

RATIONAL.

In grave cases.

Heart—the action is frequent and irregular.
Pulse—weak, small, and irregular.
Syncope.
Distended jugulars.
Restlessness and jactitation (with partial or complete pulmonic obstruction).
Delirium.
Convulsions.
Coma.

PHYSICAL.

Inspection and Palpation.—Irregular cardiac impulse; dullness to the right of the sternum.

Auscultation.—Marked irregularity of the heart's action. Murmurs, indicative of either tricuspid or pulmonic obstruction, heard with greatest intensity at the xiphoid cartilage, and conveyed to the left of the sternum.

DIFFERENTIAL DIAGNOSIS.

From lesions of the valves, or rupture of the chordæ tendineæ.

PROGNOSIS.

Is unfavorable.

TREATMENT.

Carbonate of potash, or sesqui-carbonate of ammonia; or ammon. carb. gr. xxx. every two hours, and absolute quietude.

NERVOUS CARDIAC PALPITATION.

DEFINITION.

Is a neurosis of the heart, but is not, as a rule, dependent upon heart-changes.

ETIOLOGY.

Violent physical exercise. Indulgence in alcohol.

Too rapid growth in young people, producing a narrrowing of the chest.

Debility, anæmia, excessive sexual intercourse, typhoid fever.

It may be induced by late hours, strong tea, coffee, tobacco, shock, fright, deranged digestion, flatulence, dyspepsia, gout, etc.

SYMPTOMS.

Heart—the action is irregular and often intermittent.
Palpitation—slight, quick, prolonged, or heaving pulsation.
Sense of uneasiness—in the precordial region.
Sense of constriction, weight, or pain in the region of the heart.
Sinking and fluttering sensation in the epigastrium.
Dyspnœa.
Vertigo.
Headache.
Tinnitus aurium.

PROGNOSIS.

Is good, if no organic disease exist.

TREATMENT.

Remove the cause; and assure the patient that there is no danger, provided organic disease be absent.

ANGINA PECTORIS.

DEFINITION.

Is a neurosis, dependent upon heart-changes (principally organic changes), causing defective heart-power.

MORBID ANATOMY.

It is associated with all the forms of cardiac or aortic disease, but especially with obstruction to the coronary circulation, and fatty degeneration of the muscular-tissue of the heart. It is claimed that there must be some nervous element present, having its seat in the pneumogastric nerve or cardiac plexus, causing cardiac spasm.

ETIOLOGY.

Predisposing Causes.

Obstruction to the coronary circulation from aortic regurgitation, or atheroma, or embolism of the coronary arteries.

Fatty degeneration with suddenly disturbed heart's action.

Exciting Causes.

Mental emotion.

Prolonged physical exertion.

Errors of diet.

Anything that disturbs the heart's action.

SYMPTOMS.

Rational.

Pain—intense, agonizing, stabbing, lancinating, shooting through the back and along the left arm. This is *a reflex act of the pneumogastric and phrenic or cardiac nerves to the cervical ganglia, and conveyed onward to the brachial plexus.*

Sense of suffocation.

Countenance—pale and anxious.

Face—livid, and covered with perspiration.

Pulse—faltering and almost imperceptible.

Patient is usually unable to move (on account of the severity of the pain), or even lie down.

No palpitation of the heart is usually present.

Dyspnœa—very extreme and agonizing.

Sense of approaching death.

Syncope and death—sometimes.

Physical.

Inspection.—Respiration short and hurried.

Auscultation.—A heart murmur (if the attack be due to a valvular lesion).

DIFFERENTIAL DIAGNOSIS.

From spasmodic asthma; hysteria; intercostal neuralgia; myalgia; first stage of pleurisy.

In spasmodic asthma—the physical examination will determine the absence of heart-lesions.

In hysteria—the *intermittent* and *irregular pulse* of angina pectoris will be wanting; and the pain and rational symptoms will be less severe.

In intercostal neuralgia—there will be the duration of the attack; the direction of the pain, and the absence of cardiac disturbance, to assist in the diagnosis.

In myalgia—the condition of the circulation, locality of the pain, and the physical signs, are sufficient to exclude angina pectoris.

PROGNOSIS.

Is unfavorable. The patient may recover from first attacks.

TREATMENT.

Remove the exciting cause and alleviate the symptoms. The patient must be kept quiet and *plenty of digitalis* administered; also iron, strychnia, or arsenic daily in small doses.

BASEDOW'S DISEASE.

SYNONYM.

Exophthalmic goitre—Grave's disease.

DEFINITION.

Is a disease characterized by a subjective sense of palpitation, accompanied by acceleration of the heart's action; pulsation of the veins in the neck and head; swelling of the thyroid gland; and protrusion of the eyes from their orbits.

ETIOLOGY.

The protrusion of the eye from the orbit is caused by a semi-paralysis of the vaso-motor nerves, thus affecting the blood-vessels. The muscles of the heart may be affected from the same cause (occasioning many of the other above-named symptoms). The eyes may also be caused to protrude by intra-orbital fat; by abscess, or tumor in the areolar texture of the orbit; and by exostosis of the parietes of the orbit. It may follow menstrual disorders, or lack of red blood-corpuscles in the blood. It is more common among women than men.

SYMPTOMS.

Palpitation of the heart—with remarkable frequency of pulse (120 to 140).
Swelling of the thyroid gland.
Protrusion of the eyes.
Eyelids—incapable of covering the eyeballs (which may cause infiltration or abscess of the cornea, or total destruction of the eye).
Loss of sight (sometimes) from abscess and perforation of the cornea.
Spasmodic contraction of the levator palpebræ superioris.
Peculiar rustling sound in thyroid gland (on auscultation).
Blowing sound at the heart (blood-murmurs).
Oppression in the throat.

Dizziness.
Headache.
Cyanosis.
Dropsy.
Extreme dyspnœa.
} In severe cases during the full development of the disease.

DEATH.

May occur with cerebral symptoms, or from an intercurrent disease.

TREATMENT.

Strengthening diet; *iron; secale cornutum* (ergot); *constant* and *induced current* to cervical portion of the sympathetic.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE DIGESTIVE TRACT.

Under this heading I have included:

1. Diseases of the *Mouth.*
2. Diseases of the *Pharynx.*
3. Diseases of the *Œsophagus.*
4. Diseases of the *Stomach.*
5. Diseases of the *Intestines.*
6. Diseases of the *Liver.*

The liver, being classed as one of the accessory organs of digestion, can properly be included under this head.

DISEASES OF THE MOUTH.

CATARRH OF THE MOUTH.

DEFINITION.

Catarrh is a term applied to all simple inflammations of the mucous membranes of the body; hence to the mucous membrane of the mouth.

MORBID ANATOMY.

The mucous membrane is, at first, dark-red; subsequently becoming dry, and finally a copious, cloudy secretion takes place. Early in the affection, there is swelling of the mucous membrane, increased secretions, and an excessive formation of young cells. There may be impressions of the teeth perceived upon the sides of the tongue. A mucus, which is turbid and foul, covers the cheeks, gums, and tongue (called "coated tongue").

On *microscopical examination*, the coating of the tongue consists of epithelial cells, containing fat-globules and brown granules.

SYMPTOMS.

RATIONAL.

Burning and tension in the mouth.
Slimy, clammy taste (bilious).
Foul taste or smell (often perceived by the olfactory nerve of the patient).
Craving for sour, salty, and highly-seasoned food.
In children—it may accompany teething; convulsions (reflex symptom) may also exist.

PROGNOSIS.

Is favorable, except when the attacks occur during dentition.

Unless the affection become chronic, it is usually curable in a short time, provided the patient follow the directions that are given.

TREATMENT.

Remove all irritation (as sharp edges of teeth, etc.). Forbid smoking strong cigars. When smoking is a confirmed habit, the cigars should be weak ones and a holder used. If it occur from mercurials, discontinue them. For obstinate foulness of the mouth, the patient should chew slowly small pieces of rhubarb before going to bed.

APHTHÆ.

SYNONYM.

"Croupous stomatitis"—a frequent disease of infants.

MORBID ANATOMY.

It occurs on the anterior half of the tongue and inner surface of the lips, cheek, and hard palate, in gray or yellowish-white deposits with a red border, which excoriates the parts, but does not cause ulceration. On the free surface of the mucous membrane under the epithelium, are small solid white spots with a red border (consisting of exudations).

ETIOLOGY.

It occurs chiefly in weakly and badly-nourished children, or, may be due to dentition. It may also occur as an epidemic.

SYMPTOMS.

Fever.
Restlessness.
Loss of appetite.
Pain—which is increased when the child is being suckled.
Fetor of the mouth—from decomposition of the epithelium and the exudation which is thrown off.

TREATMENT.

Chlorate of potash, gr. iv.-vi. should be given, or the parts painted with dilute muriatic acid, or a solution of *nitrate of silver* (gr. i. to ʒ ss. of water).

CANCRUM ORIS.

SYNONYM.

"Diphtheritic stomatitis."

DEFINITION.

Is a fibrinous exudation, accompanied by sloughing of the mucous membrane, from compression of the vessels (due to exudation).

MORBID ANATOMY.

In mild cases—it shows itself in the form of white spots along the lateral border of the tongue and on the parts of the cheek and lips which lie against the teeth. These spots after a time fall off and leave an ulcer.

In severe cases—when the whole mucous membrane is destroyed, a large portion of the mouth is converted into a soft discolored slough which may separate and leave an irregular border and an uneven base. This may be replaced by cicatricial tissue. Ad-

hesions and false anchylosis may occur, and sometimes caries and necrosis of the maxillary bones may ensue.

ETIOLOGY.

From excessive use of mercurials. It occurs as an epidemic in Foundling Hospitals, Orphan Asylums, Barracks, Armies in the field, etc.

SYMPTOMS.

Severe pain—especially when there are ulcers formed.
Increased salivary secretion.
Insomnia—due to the secretions going into the larynx and producing fits of coughing, or suffocation.
The edges of the teeth are coated with a foul deposit.
Fetid odor to the breath.
Gums—dark, red, and swollen from excessive hyperæmia.
Teeth—loosened and bleed easily.
Diphtheritic pulpy deposit on the gums.
Lymphatic glands are swollen and painful.
Œdema of the lips and cheeks.
Saliva—bloody and discolored.

PROGNOSIS.

When properly treated, it runs a favorable course and heals.

If it be neglected, it may last for months. Is fatal only when complications occur.

TREATMENT.

The cure is slow. It will take eight to nine days to get the patient even in a comfortable state. Wash the mouth out with cold water, or water and red wine, or *chlorate of potash* ℈ j-ʒ j-ij dissolved in ℥ vi. water. Paint the mouth with a solution of *nitrate of silver* (gr. i.-℥ ss.), or touch the ulcers with solid nitrate of silver. This last treatment is exceedingly painful.

THRUSH.

SYNONYMS.

"Soor," "muguet," or "sprue."

MORBID ANATOMY.

There is a whitish, delicate, frosty coating on the inner surface of the lips, tongue, and roof of the mouth, advancing to the larynx and œsophagus. It may extend, downward, along the digestive tract.

ETIOLOGY.

From fungus growing on the mucous membrane of the mouth; diminished secretions of the mouth; and the neglect of cleansing the mouth. It occurs most often in infants; and in adults, generally before death. It is always a bad symptom in adults if following some serious disease.

SYMPTOMS.

Painful burning in the mouth.
(*In a child.*)
Diarrhœa—with pain in the abdomen.
Stools—fluid, green, and acid.
Anus—red, and excoriated.

TREATMENT.

Cleanliness of the mouth; which must be washed out with wine and water after feeding. *Borax* is often used as a local application.

NOMA.

SYNONYMS.

"Water-canker," or "gangrenous sore mouth."

MORBID ANATOMY.

It commences on the inside of the cheeks, over a hard spot which has previously developed; the mucous membrane is red, and discolored, and vesicles filled with serum form on this spot. The part becomes black, softens, and disintegrates; the gums are destroyed, and also the lips and base and edges of the tongue on the affected side. The teeth become loose and fall out. The whole of one side of the face may have become a black slough (in

about five or six days). On *post-mortem* examination, the blood-vessels are filled with a fibrinous coagula.

SYMPTOMS.

Œdema—of the affected cheek and lips, with a hard, round nucleus at the seat of the disease.
Saliva—is bloody and black.
Pulse—small and frequent.
Delirium—at night.
Thirst—unquenchable.
Diarrhœa—is usually present.

PROGNOSIS.

Recovery is rare; and, when it does occur there is a large cicatrix left, often causing fearful disfigurement.

TREATMENT.

Give the patient plenty of fresh air, good nourishment, wine, etc., and treat the gangrene locally by actual cautery.

SYPHILITIC AFFECTIONS OF THE MOUTH.

VARIETIES.

Primary--often due to nursing of an infant by a syphilitic nurse; the affection may appear during later years in the form of ulcers and condylomata.
Secondary—occurs in the early stages, and both forms (ulcers and condylomata) often appear together.
Tertiary—occurs in the later periods, in the form of gummy tumors, or nodular syphilomata. Caries of the hard palate may also be developed.

MORBID ANATOMY.

The primary and secondary ulcers and condylomata spring from circumscribed indurations, or syphilitic papules of the mucous membrane, giving a white appearance to the affected part. They

occur especially at the corners of the mouth, and have the appearance as if the corners were torn. Condylomata usually form on the lateral edges or dorsum of the tongue. Gummy tumors frequently appear on the anterior third of the tongue which soon soften and rupture, leaving deep ulcers.

SYMPTOMS.

Pain—on chewing, and sometimes of mouth.

Mucous patches—these often disappear and reappear on some other spot.

Tongue—is unwieldly, and often rigid if extensively diseased.

TREATMENT.

Mercury and *iodide of potash*. *Calomel* as a dusting powder to the condylomata. *Fumigation* of the mouth *with* the *vapor of calomel* or the *black oxide of mercury*.

ULCERS OF THE MOUTH.

MORBID ANATOMY.

There may be small vesicles at the end of the tongue, and ulcers showing catarrhal inflammation. Variolous ulcers may appear on the roof of the mouth, and herpetic ulcers may occur on the inside of the cheek; the bases of these ulcers have a yellow or lardaceous appearance, and their edges are usually elevated, hard, and red. Syphilitic ulcers (mucous patches) are most frequent in the mouth or fauces. There may be small excoriations, as if the mouth had been burnt, from smoking, or drinking something hot.

ETIOLOGY.

Follicular ulcers from stoppage, swelling, and ulceration of mucous glands, may occur at the menstrual epoch, or during pregnancy, or lactation. The other types depend upon special blood conditions.

SYMPTOMS.

Great pain—when the ulcers are diffused and not syphilitic.

Disturbed digestion—(may occur) if the ulcers be follicular in character.

PROGNOSIS.

Is rarely dangerous, although the ulcers may last for weeks. If syphilitic, they recur and tend to progress forward toward the lips.

TREATMENT.

Smoking, drinking hot beverages, eating highly-seasoned food, etc., must be avoided. The ulcers should be touched with solid nitrate of silver. A weak solution of corrosive sublimate will often be found very beneficial.

SALIVATION.

SYNONYM.

"Ptyalism."

DEFINITION.

Is a disease where the increased secretion of the salivary glands ceases to pass into the stomach with the ingesta; but flows constantly out of the mouth, if the case be severe.

MORBID ANATOMY.

The saliva contains young and old epithelial cells. The saliva is alkaline, and contains fat and ptyaline, and sometimes sulphocyanide of potassium and mucus corpuscles. The teeth become loose, if due to mercury; the tongue is oftentimes so swollen as to protrude; while the peculiar fœtor of mercurial is very marked.

ETIOLOGY.

Simple salivation may occur from irritation of the mucous membrane of the mouth, or pharynx; from introduction of irritating substances into the mouth; from irritation of the lingual branch of the trifacial, or glosso-pharyngeal nerve; from neuralgia of the trifacial nerve; from irritation affecting the gastric, or intestinal mucous membrane, or uterus, or other organs; from

mental influences (disgust and desire); from pregnancy; from disease, as typhus or intermittent fever, etc. The most marked type of salivation follows mercurial poisoning.

SYMPTOMS.

Constant spitting—(at the onset).
Emaciation.
Constant flow of saliva—sometimes reaching from six to eight pounds in twenty-four hours.
If due to mercurial poisoning—a swollen and protruding tongue; loosened teeth; fœtid breath.

TREATMENT.

If it occur from misuse of mercury, give slight laxatives and purgatives, and an astringent gargle.

If from affections of the stomach, etc., then treat the disease.

General baths, blisters, mustard to the throat and nape of the neck, mouth washes of *alum, sulphate of zinc, sage, or oak-bark* have been recommended, as also *opium,* to diminish the excitability of the nerves.

PAROTITIS.

SYNONYM.

"Mumps."

VARIETIES.

Idiopathic; symptomatic; polymorpha or "mumps;" or metastatic.

MORBID ANATOMY.

Idiopathic parotitis—Is believed to be due solely to a serous exudation from a catarrh of the gland-duct, affecting the interstitial substance and connective-tissue about the gland.

Symptomatic parotitis—Begins with hyperæmia, causing infiltration and swelling of the interstitial substance of the gland. The gland becomes tough and filled with a whitish or yellowish substance, which soon turns purulent, giving the gland the

appearance of lobules filled with pus. Destruction of the tunica propria takes place, and suppuration of the interstitial-tissue occurs, which extends rapidly, and often results in a large parotid abscess. Sometimes there is destruction of the gland-tissue, and gangrene attacking the neighboring connective-tissue, and muscles of mastication, and also the periosteum of the maxillary, temporal, and sphenoid bones, and sometimes the bones themselves. The inflammation, in severe cases, often extends to the brain, or internal and middle ear; this may take place along the blood-vessels and nerve-sheaths, as well as the bones themselves. It sometimes induces phlebitis and thrombus of the neighboring veins (especially of the anterior and posterior facial and external jugular) and disintegration of these thrombi, thus producing embolism and septicæmia.

SYMPTOMS.

Idiopathic form.

General febrile symptoms for two days.

Swelling—near the lobe of the ear, extending to the cheek and neck; and then to the other side of the face. After five or six days, the fever ceases and all the symptoms may disappear in ten days. The tumor may become very painful, hard, and dark-red; abscesses may form, which open outwardly, or into the external auditory meatus

Testicles—may be affected (by metastasis).

Pain—in the sacral and inguinal region may exist and an exacerbation of the fever may occur.

Œdema—of the scrotum (in men), and vulva and breast (in women), and pain in the ovaries.

Meningitis—may sometimes occur and prove fata..

Symptomatic form.

Occurs in various diseases, and is accompanied by a chill, or exacerbation of fever. It usually affects only one side.

When suppuration takes place, the swelling becomes uneven, nodulated, and red. Fluctuates at points; may open outwardly into the meatus auditorius externus, but generally into the mouth or pharynx; or it may burrow

down into the œsophagus, trachea, or chest. While mortifying, the skin becomes dark-blue and discolored, doughy in feel, and depressed. On opening tumor there is evacuated a discolored pus with shreds of tissue.

TREATMENT.

In the idiopathic form.—The bowels and digestion should be regulated, and the patient protected from all injurious influences. The part should be covered with wadding, etc. Much meat should not be allowed, only food which is easily digested. If the tumor be hard and swollen, leeches should be applied; if fluctuant, it should be opened and poultices applied.

In the symptomatic form.—If the swelling be red or painful on pressure, apply ice and cold water; if fluctuant, open and apply poultices.

GLOSSITIS.

DEFINITION.

Is a parenchymatous inflammation of the tongue.

VARIETIES.

Acute—Chronic—Dissecting—and Superficial or "psoriasis."

MORBID ANATOMY.

In the acute form.—The whole tongue becomes very large, darkened in color, smooth, or fissured, and covered with tough bloody exudations. Abscesses may form and leave a scar.

In the chronic form.—Only a part of the tongue may be affected, especially the edges, where circumscribed spots may project.

In the dissecting form.—The tongue is divided into lobules by furrows, causing ulceration from lodgment of food, etc.

In the superficial form.—The tongue looks glossy and smooth as though it were varnished or scalded.

SYMPTOMS.

Tongue—is enlarged and projects often one inch beyond the teeth, and is white or dirty-brown on its upper surface, or dark-red

on the under surface; ulcers may occur, or there may be impaired movements and the speech unintelligible.

Flow of saliva—is constant.

Glands of the neck are enlarged.

Face may be blue and swollen from obstruction to the jugulars.

Impaired respiration—from stoppage of entrance of air into the larynx, due to swelling of the tongue backward, may occur in severe cases.

In the acute form.

High fever.

Full pulse.

Anxiety.

Restlessness.

Asphyxia.

In the chronic form.

Circumscribed dull pain, which may last for years.

TREATMENT.

In the acute form.—Scarify deeply. Then put ice in the mouth and give soothing washes. If suffocation threaten, then perform tracheotomy.

In the chronic form.—Remove all obstacles to recovery. Should this fail, an operation must be resorted to.

In the dissecting form.—Treat the ulcer with *nitrate of silver* in substance or solution.

In the superficial form.—Do not give mercury, as it increases the pain, but rinse the mouth out with a dilute solution of carbolic acid, and touch the fissures with pure carbolic acid.

TONSILLITIS.

SYNONYM.

"Quinsy."

DEFINITION.

Is an inflammation of the interstitial-tissues of the tonsils, and proliferation of the connective-tissue, which may be acute or chronic in character.

MORBID ANATOMY.

The tonsils sometimes swell to the size of a walnut, the surface is nodulated, dark, and red, and covered with a glutinous exudation, or croupous deposits. Suppuration may occur.

SYMPTOMS.

Chill and high fever (especially if pus be developing).
Temperature—104°, and sometimes even higher.
Pulse—frequent and full.
Throat—tense and sore.
Pain—piercing, and shooting toward the ear.
Difficulty and pain in opening the mouth, or in chewing.
Mouth—cannot be opened widely.
Respiration—is affected.
Uvula—may be deflected to one side, and tonsils enlarged.
There may be severe headache.
Insomnia may be present in severe cases.
Delirium may be present in severe cases.
Symptoms of asphyxia—may occur, if extensive suppuration occur.

TREATMENT.

Powdered alum (two or three times daily) should be applied to the affected part. Rinse out the mouth with a solution of alum, ʒ iij.–ʒ ss. to ʒ vi. of barley-water. Apply *nitrate of silver* to the affected part. *Cold applications* may be used—as ice, cold water, or cold compresses, changing them frequently. If fluctuation occur, a warm poultice should be applied, and the mouth washed out with *chamomile tea*. Open the abscess early with the finger-nail, or lancet. Be careful in using the knife, as the carotid artery may be wounded.

If there be cerebral troubles, give laxatives. If it become chronic, paint the parts with a *solution of alum, nitrate of silver, dilute tincture of iodine;* or cold compresses may be applied.

ANGINA LUDOVICI.

DEFINITION.

Is a phlegmonous inflammation of the floor of the mouth, and intermuscular and subcutaneous connective-tissue of the submaxillary region, leading to gangrene and sloughing, or ending in abscess, or resolution. It is often termed "Gangrenous inflammation of the neck."

ETIOLOGY.

From periostitis of the lower jaw, or metastatic parotitis, occurring in typhus and other infectious diseases, starting from the submaxillary gland.

SYMPTOMS.

Swelling in the floor of the mouth (upward and downward).
Loss of motion of the tongue.
Inability to open the mouth (abscesses may form).
Fever and general constitutional derangement.
Death—from œdema glottidis, suffocation, or septicæmia, etc.

TREATMENT.

Apply a large number of leeches near the seat of the tumor. When the tumor is fluctuating in character, the contents should be evacuated as soon as practicable. If suffocation be apprehended, then scarify. Tracheotomy may be resorted to in dangerous cases. If a hard tumor remain for a long time, then apply blisters freely and repeatedly.

DISEASES OF THE PHARYNX.

PHARYNGEAL CROUP.

DEFINITION.

Is a fibrinous inflammation of the pharyngeal mucous membrane.

MORBID ANATOMY.

White, or grayish-white membranous masses are seen on the reddened mucous membrane of the soft palate, tonsils, and pharynx, like small irregular round islands. The membrane often adheres so firmly that, on detaching it, a bloody superficial loss of substance remains. This indicates a change from the croupous to the diphtheritic form.

SYMPTOMS.

Dryness of the throat, local inflammation, and gray patches—which may be mistaken for ulcers.
Great pain—in the arches of the palate, chiefly on attempts to swallow.
Uvula—elongated.
Nasal tone of voice.
Coated tongue, etc.
Pulse and temperature—may be elevated, if the case be severe.

TREATMENT.

If the attack be mild, it should be left alone; but if of a severe type, apply *moist compresses* well wrung out, and covered with dry cloth, or warm poultices, to the throat. Wash out the mouth with water, or a *solution of alum, sulphate of zinc, acetate of lead*, etc. Cover the inflamed parts with powdered alum, or paint them with a solution of *nitrate of silver* (ʒ i. to ʒ i. water). If it accompany croupous laryngitis, remove the membrane, and cauterize it with nitrate of silver.

RETRO-PHARYNGEAL ABSCESS.

DEFINITION.

Is an abscess between the textures of the pharynx (its posterior wall) and the cervical vertebræ.

MORBID ANATOMY.

The posterior wall of the pharynx is pressed forward by pus,

causing contraction, or closure of the pharynx. It may perforate the pharynx, or even extend down to the pleura.

SYMPTOMS.

Difficult swallowing.
Restlessness.
Croupy coughing and choking.
Continual dyspnœa.
Large fluctuating tumor.

CAUSES OF DEATH.

Complete closure of the glottis.—Œdema glottidis.—Opening of the abscess during sleep.—Entrance of its contents into the larynx.—Pleuritis.—Pneumonia.—Pericarditis.

TREATMENT.

Open the abscess at once with the finger-nail, or lancet.

DISEASES OF THE ŒSOPHAGUS.

STRICTURE OF THE ŒSOPHAGUS.

It occurs most frequently in the lower third, but it may affect any part of the tract. The walls are hypertrophied and the canal dilated above the stricture; while the walls are thinned and the canal collapsed below the stricture.

MORBID ANATOMY.

It depends on cicatricial contractions and considerable loss of substance (as in corrosions or ulcerations) or, on hypertrophy of the muscular, or intermuscular connective-tissue; from chronic catarrh (which on section gives a fan-like appearance) or, from hypertrophy of the submucous-tissue.

ETIOLOGY.

From compression due to swelling of the thyroid body; swelling of the lymphatics; dislocation of the hyoid bone; exostosis of

the vertebræ; abscess; tumor; aneurism; carcinoma of the lungs; dilatation of the right subclavian artery; protrusion of new growths into its canal (carcinomatous); structural changes in its walls; previous inflammation and ulceration.

SYMPTOMS.

Difficulty of swallowing.
Pain about the manubrium and back.
Regurgitation of food.
Constipation from starvation.

TREATMENT.

Dilatation—by mechanical means.

DILATATION OF THE ŒSOPHAGUS.

MORBID ANATOMY.

In total dilatation.—The canal may be the size of a man's arm, with hypertrophied or thinned walls.

In partial dilatation.—That portion of the canal above the constriction is usually the largest.

The diverticuli usually form near the bifurcation of the trachea, or where the pharynx terminates, and the œsophagus begins.

ETIOLOGY.

It may be *partial* when limited to a short distance, or total when the entire organ is affected. When partial, only one wall may be affected. Localized enlargements may occur, developing into large sacs, communicating with the œsophagus, and termed diverticuli; these (diverticuli) are formed by foreign bodies that have stuck into the walls, and have been driven further in by the food as it passes down, or, by the shrinkage of the bronchial glands which were adherent, when swollen, to the mucous membrane, and, on contracting, draw the mucous membrane after them. *Total dilatation* is due to a chronic catarrh, which induces muscular paralysis.

SYMPTOMS.

In partial dilatation.

Retention of food—then regurgitation (the food being mixed with mucus). The food may sometimes be decomposed.

Diverticuli—when present may be felt in the neck like a soft tumor.

Dyspnœa—from the diverticuli causing compression of surrounding structures (as the pneumogastric, laryngeal, or phrenic nerves).

Death—from starvation.

TREATMENT.

Is of no use. The stomach tube may be resorted to, provided it can be passed through the diverticuli. In this event, there may be some slight hope of recovery.

PERFORATION AND RUPTURE OF THE ŒSOPHAGUS.

ETIOLOGY.

May occur from within outward or the reverse.

From within outward.

From breaking down of a cancer; ulcer (caused by splinters of bone (rare)); deep sloughs (excited by corrosion).

From without inward.

From aneurism of the aorta; breaking down of a tuberculous gland at the bifurcation of the trachea; abscesses in the interior surface of the spine; caries of the vertebræ; tuberculous cavities in the lungs.

SYMPTOMS.

Pain—severe and sudden, in the breast.

Chill.

Paleness.

Coldness of the extremities.

Faintings and attacks of suffocation.

Profuse vomiting of blood (in case of aneurism), or of pus (in case of abscess).

TREATMENT.

None.

NERVOUS AFFECTIONS OF THE ŒSOPHAGUS.

Globus Hystericus.—Hyperæsthesia, or increased excitability of the sensory nerves, and feeling as if the œsophagus were ligated, and a supposed inability to swallow.

Hyperkinesis or *Dysphagia Spastica.*—Is an increased excitability of the motor nerves. The spasm is reflex and forms a symptom of brain-disease, or of the upper part of the cord; or, it may result from poisoning by narcotics or in alcoholism. It usually occurs during eating.

Akinesis.—Is a diminished excitability of the motor nerves. It accompanies diseases of the brain and cervical portion of the cord. If complete paralysis exist, there is inability to swallow; but if incomplete paralysis, large pieces of meat and food are able to be swallowed.

TREATMENT.

For Globus hystericus or Hyperkinesis.

Belladonna, valerian, asafœtida, musk, etc. Also repeated and careful introduction of the bougie.

For Akinesis.

Probe, electricity, and strychnia (?).

DISEASES OF THE STOMACH.

ACUTE GASTRIC CATARRH.

DEFINITION.

Is an acute catarrhal inflammation of the mucous membrane of the stomach.

MORBID ANATOMY.

The mucous membrane is reddened in spots, but the result is generally negative. Beaumont's observations on St. Martin showed the mucous membrane reddened and covered with tough mucus, with traces of blood; the mucous covering became subsequently thickened, and the gastric juice suppressed. The walls of the stomach, *on post-mortem*, may show spots of softening, and the mucous lining may be eroded from fermentation.

ETIOLOGY.

It occurs in fevers, pneumonia, etc. (from neglect of simple dietetic rules, producing anorexia, and capricious appetite); in debilitated and badly nourished persons (producing diminution in the gastric juice from decreased amount of blood in the system and hydræmia resulting in a diminution of albuminates, of which pepsin is made and which is the organic constituent of the gastric juice). From excess in drinking and error in diet in children: large quantities of easily digested food taken, and diminution of gastric juice causing decomposition of food; use of indigestible food (producing decomposition); substances that have commenced to decompose before entering the stomach; irritation of hot or cold articles, medicines, alcohol or spices; introduction of articles that weaken the gastric juice, or retard the movement of the stomach (producing abnormal decomposition of its contents), as misuse of alcohol, etc.; from narcotics (producing impaired movements of the stomach); catching cold; and infection.

SYMPTOMS.

General malaise and feeling of nausea.
Dullness and fretfulness.
Head—hot, and possible headache.
Extremities—cold.
Pressing pain in the forehead, extending to the occiput.
Flashes before the eyes on stooping.
Pain at the pit of the stomach.
Distaste for food and great thirst.
Belching of sulphuretted hydrogen and carbonic acid gas.

Sour fluid occasionally rises in the mouth.
Tongue—coated.
Taste—stale and slimy
Retching and vomiting.
Severe diarrhœa.

PROGNOSIS.

Previously healthy persons rarely die. From repeated attacks, weakly and decrepit persons may die of gastric or catarrhal fever. *In children*, it may end fatally.

TREATMENT.

Watch the diet carefully. Milk should be the principal diet. If injurious food has been taken, give an emetic of *ipecacuanha* or *tartrate of antimony.*

Should there be prominence of the epigastrium and eructation of gases, give ipec. ℈i. and tart. antimony gr. i., but not where the fever is high and there are signs of typhus fever present. If colicky pains, escape of flatus, etc., exist, administer rhubarb or compound infusion of senna or magnesia usta (℥ ss. to ℥ viij. of water) one tablespoonful every hour or two. If it should occur from excess of beer or wine, *sodii bicarb.* gr. v.–x. should be given. If the catarrh be slight and there be characteristic vomiting, give pulv. rhei co.; if diarrhœa be more severe, tr. rhei aquosa, or small doses of calomel (gr. $\frac{1}{8}$–$\frac{1}{4}$) should be given two or three times daily, or ℞ calomel, gr. iv.; pulv. jalapæ, gr. ij.; sac. albi, ʒ ss.; fiat pulv. viij. Take one powder in water every two hours; or ℞ argent. nit., gr. $\frac{1}{4}$; aquæ, ℥ ij. M. Sig. ʒ i. every hour. If the catarrh be slight and no vomiting exist, ℞ acidi tannici, ℈ss.; aquæ, ℥ iij. M. Sig. ʒ i. every two hours, may be administered.

CHRONIC GASTRIC CATARRH.

MORBID ANATOMY.

The mucous membrane is a reddish-brown, or slate color, from transformation of the hæmatin into pigment. There is a coarse anastamosis, or dilatation of the vessels. The mucous membrane

becomes hypertrophied and forms numerous folds. There are innumerable small prominences, separated by furrows, which are most frequent in the pyloric end of the stomach. A grayish-white tough mucus covers, and clings to, the inner surface of the stomach; and, constriction of the pylorus takes place, through thickening of the walls of the stomach, from simple hypertrophy.

ETIOLOGY.

From continued and repeated attacks of the acute form; habitual misuse of spirituous liquors (especially); congestion of the gastric mucous membrane; obstructed circulation in the portal vein or liver. It accompanies phthisis and other chronic diseases: cancerous or other degenerations of the stomach; and may extend to the mouth and intestines.

SYMPTOMS.

Abnormal sense of pressure and fullness in the stomach (which is increased by eating).
Prominence of the epigastrium—(from gas).
Constant eructations—(from stricture of the pylorus).
Vomiting (rare)—occurs in drunkards as "water-brash."
Sensation of hunger is lost (in some cases).
Wolfish appetite (in some cases).
Mental depression.
Change in the urine—(difficult to understand) becomes scanty.
Death—may occur from marasmus (due to stricture of the pylorus or dropsy).

PROGNOSIS.

Is rarely fatal.

TREATMENT.

Never give emetics. Forbid all spirituous liquors. Patient should wear warm clothing, and take baths to excite the activity of the skin. Do not allow a vegetable diet, fat meats, or sauces. The diet should consist of lean meat which must be slowly and carefully masticated, and only small quantities to be taken at a time, or cold meat and a little white bread, or salt or smoked meat and sea-biscuits; milk, or butter-milk which is better (ad libitum).

Soda or Karlsbad water may be prescribed, or bismuthi nit. gr. x. Argenti nit. gr. i.-ij. before breakfast (while fasting).

If there be atony of the gastric mucous membrane, give *iron* and *mild stimulants*, also ipec. gr. ss.-ij.; pulv. rhei gr. iij.-iv. in pills before meals. If constipation be present, give an enema, or some laxative such as aloës, ext. colocynth co. or comp. ext. rhei. Aloes and colocynth are the best.

Never administer senna or oleum ricini.

CARCINOMA OF THE STOMACH.

MORBID ANATOMY.

The cancer may vary in size from about the size of a pigeon's egg to that of a man's fist; it attacks the pyloric portion, principally, and spreads transversely, causing an annular stricture. The most frequent form of cancers are the scirrhus, medullary, alveolar or colloid (rarest). The colloid lasts a long time; induces ascites; and after tapping, may be felt as nodular masses appearing on the omentum.

Scirrhus—begins in the sub-mucous tissue, forming irregular nodules; it has the appearance of a dull, whitish, dense mass of cartilaginous hardness, which softens to a black pulp and then sloughs.

Medullary—looks like brain substance; on section giving off a "cancer juice." It spreads more rapidly than the other forms (often reaching the size of the hand).

Alveolar or Colloid—occurs more frequently as diffused degeneration. Commences in the submucous tissue and contains a gelatinous fluid. It often extends to other organs, especially the lymphatics, pancreas, liver, transverse colon, or omentum. It induces degeneration of the peritoneum and consequent ascites. Prior to adhesion to other organs, the contents of the stomach may escape into the abdomen, if the ulceration penetrate its coats.

ETIOLOGY.

Is obscure. It is sometimes primary, but usually secondary, to cancerous degeneration of other organs. It may be hereditary,

and occurs more in men, especially between forty and sixty years of age.

SYMPTOMS.

Emaciation.
Feeling of pressure in the epigastrium.
Patient becomes apathetic.
Loss of appetite, and constipation
Vomiting.
Tumor in the epigastrium—(sometimes absent to the touch)—most of those that can be felt are at the pyloric end of the stomach, and hence the tumor is generally on the left of the median line of the body, or near the umbilicus, from sinking of the pyloric end; it is uneven, nodular, and movable, unless there be adhesions.
Dirty-yellow cachectic color of the skin.
Œdema of the ankles.
Tenderness in the region of the stomach—which is increased by pressure, and after eating.
Coffee-ground vomiting—resulting from capillary hemorrhage, caused by breaking down of the vascular growth. The effused blood is altered by the acid contents of the stomach into this black coffee-like mass.
Hard swelling of the supra-clavicular glands, (may be diagnostic).

CAUSES OF DEATH.

From exhaustion (when, prior to death, the tongue becomes red and dry, and aphthous deposits occur on the tongue); œdema of the legs ensues from obstruction to the femoral vein, showing that a clot has formed; frequently from peritonitis (due to rupture of the stomach); from complications; and secondary diseases.

DIFFERENTIAL DIAGNOSIS.

1. From chronic catarrh of the stomach—but the tumor in the epigastrium, and coffee-ground vomiting decides it; and there is also the general condition of the patient to assist in the diagnosis.
2. From chronic ulcer of the stomach.

TREATMENT.

Milk should be taken freely if it can be tolerated, or broth. The yolk of an egg (always in small quantities) and red wine. For acidity of the stomach, give soda water. *The best method of administering food or other stimulants is by the rectum,* as the stomach is thus at rest, and irritation greatly relieved.

NERVOUS CARDIALGIA.

DEFINITION.

Is a painful affection of the stomach, which is not dependent upon any perceptible changes in the structure; but upon hyperæsthesia of the pneumogastric nerve, or solar plexus of the sympathetic.

ETIOLOGY.

It occurs in anæmic persons and chlorotic women (from poverty of blood); from diseases of the uterus - such as dislocations, flexions, chronic inflammation, follicular ulcers of the os uteri, and inflammation of the ovaries occurring at the period of menstruation; from diseases of the brain or spinal cord; from organic changes of their pneumogastric or sympathetic nerves; from swelling of their neurolemma, or tumors pressing upon them; from dyscrasia; from excessive acidity, worms, certain medicines, beverages, etc.

SYMPTOMS.

Paroxysms of severe pain.
Peculiar feeling of pressure in the stomach.
Severe griping pain in the pit of the stomach, extending to the back.
Feeling of faintness.
Countenance becomes shrunken.
Feet and hands become cold.
Pulse—small and intermittent.
Epigastrium—puffed out, or sometimes retracted (with tension of the abdominal walls).

Epigastric pulsation.
Pains in the thorax (sympathetic) under the sternum.
Exhaustion—severe (after the attack).
Eructation of gases and watery fluid (after the attack).
Urine—red color and scanty.

DIFFERENTIAL DIAGNOSIS.

It may be confounded with ulcer of the stomach.

PROGNOSIS.

Is favorable, unless complicated by some incurable disease.

TREATMENT.

For those suffering from anæmia or chlorosis, *iron* should be administered at once in either of the following forms: Ferri carbonas sacch. (British), gr. iv.–x.; tinc. ferri chlor., gr. x.–xxx.; ferri sulph., gr. i.–iv. or *Bland's pills*, which consist of the following ingredient: ℞ Ferri sulph. pulv.; potass. carb. puræ, āā ʒ ss.; tragacanth, q. s. M. fiat pil. xcvi. Take 3 pills three times a day.

Should this affection occur from malaria, *quinine* should be given.

DYSPEPSIA.

DEFINITION.

Is an impaired state of digestion, arising without any perceptible change in the structure of the stomach.

ETIOLOGY.

It may arise from restricted, too scanty, or abnormally increased secretions. It occurs in the aged from lack of material to form the necessary secretions, or from diminished excitability of the gastric nerves. Over-feeding; too little exercise; excessive study; emotional excitement; over-indulgence in spirits, tobacco, etc., may induce it.

SYMPTOMS.

Loss of appetite.
Flatulence.

Nausea—(sometimes).
Eructations—(bitter or acid).
Tongue—furred, flabby, large, or indented at the sides.
Headache.
Pyrosis.
Heartburn.
Constipation.
Hypochondriasis.
Palpitation of the heart.

TREATMENT.

This consists principally in regulating the diet and slight medications. The meals should be regular and *never hurried over;* they should be of a plain character, and easy of digestion. The food should be properly masticated and the stomach should not be over-crowded with food; "*it should be taken sparingly.*" Exercise in the open air daily is of the utmost importance. Should tonics be required, *quassia,* or *colombo,* are the most reliable stomachics. In order to overcome constipation, *rhubarb* may be given alone, or, with *extract of colocynth, aloes,* or *podophylli resina;* or any of the mineral waters may be used. *Bicarbonate of sodium* (gr. v.–x.), or *bicarbonate of potassium* (gr. ij.–v.), or *lime water* (℥ ij.), will be found of great service in case of acidity. Should there be any derangement of the liver, a blue pill at the onset of the affection may often prevent further trouble.

HEMORRHAGE FROM THE STOMACH.

SYNONYM.

Hæmatemesis.

MORBID ANATOMY.

On *post-mortem* examination, there is a superficial softening of the stomach from capillary hemorrhage, also superficial excavations in the stomach (called hemorrhagic erosions). The mouth of the vessels may be gaping. There are red clotted masses of blood in the stomach if death be sudden, or there are brown or

black clotted masses, as if the blood had escaped slowly and been acted upon by the gastric juice.

ETIOLOGY.

From rupture of over-filled blood-vessels; from arterial fluxion, causing rupture (rare); from venous congestion of the gastric mucous membrane; from impediment to the circulation of the liver (portal) by blood-clots; from pressure due to cirrhosis of the liver, or enlargement of the gall-ducts; from destruction of the capillaries by yellow atrophy of the liver; from rupture of diseased vessels; from rupture of varices and aneurisms; from exhaustive diseases, as yellow fever, scurvy, typhus, etc.; from improper living, as abstaining from meat and vegetables; from erosion or injury to the walls of the stomach, as chronic ulcer, cancer, etc.; from corrosive substances; from sharp instruments; from blows and kicks; from swallowing of blood in epistaxis and hæmoptysis; from miliary aneurism of the gastric artery.

SYMPTOMS.

Blood mixed with vomited matter.
Abnormal sense of pressure about the stomach, and a desire to loosen the clothes.
Feeling of constriction and nausea.
Paleness.
Tinnitus aurium.
Cold skin (if the hemorrhage be profuse).
Pulse—small (if the hemorrage be excessive).
Dizziness.
Fainting—(momentary fainting arrests the hemorrhage and favors the formation of coagula).
Nausea—accompanied by a sweet taste in the mouth and by a feeling of warm fluid rising in the œsophagus.
Violent vomiting of blood—partly fluid and partly clotted.
Passage of blood from the bowels—(black and clotted, or black and tar-like).
Hydræmia and dropsy—from loss of blood.

DIFFERENTIAL DIAGNOSIS.

Between hæmoptysis and hæmatemesis.

PROGNOSIS.

Favorable, unless complications exist.

TREATMENT.

If the portal circulation be obstructed, *leeches* should be applied to the anus, or os uteri. Cold ice-water should be drank, or ice eaten, and the epigastrium covered with cold compresses, or ice. The patient should lie flat in the bed. If there be syncope, let the patient inhale ammonia or eau-de cologne through the nose, or throw cold water on the face, and give cold champagne to drink.

CHRONIC ULCER OF THE STOMACH.

VARIETIES.

See page 12.

MORBID ANATOMY.

It occurs in the stomach, or upper part of the duodenum, generally near the pyloric orifice, and on the posterior wall. It has the appearance of a circular hole (like a terrace) extending from the sub-mucous coat toward the serous, with sharp borders as if a piece had been punched out. The ulcer is from about a quarter to half an inch in diameter usually, and may be round, elliptical, or bulging.

ETIOLOGY.

Disease of the walls of the vessels of the stomach; poverty of the blood and chlorosis; acute or chronic catarrh of the gastric mucous membrane. Long continued pressure, as exists in sewing girls, from their position.

SYMPTOMS.

Peritonitis—by perforation.
Hæmatemesis—by erosion of the large vessels.
Oppression in the epigastrium—which is increased by eating.
Pains in the epigastrium—which are steady and increased on

pressure at one spot. This pain extends toward the back (called cardialgia) and occurs soon after meals. *If the pains occur directly after eating, the ulcer is near the cardiac orifice. If the pains occur one or two hours after eating, it is near the pyloric orifice.*

Vomiting—this, like the pain, indicates the position of the ulcer. Severe cardialgia and vomiting usually occurs directly after meals. If blood exist, it shows the presence of a chronic ulcer. Severe cardialgia may also arise from adhesion of the stomach to some neighboring organ, impeding its motion.

Tongue—red and furrowed.

Increased thirst.

Habitual constipation.

Cachectic look and great debility.

CAUSES OF DEATH.

Death may occur from perforation of the walls and escape of the contents of the stomach into the abdominal cavity, with sudden pain, cold skin, small pulse, sunken countenance, collapse, and cyanosis (indicating paralysis of the sympathetic system); from hemorrhage of the stomach (rare); from erosion of a large artery; or from gradual exhaustion, even when the ulcer has healed.

DIFFERENTIAL DIAGNOSIS.

From chronic gastric catarrh, where it runs its course without any pathological symptoms, and from stricture of the pylorus.

PROGNOSIS.

Is on the whole favorable.

TREATMENT.

The diet should be liquid in character, consisting of milk, bread and milk, sour milk, essence of beef, or Trommer's malt extract. No vegetables should be allowed. Karlsbad or warm mineral spring waters may be prescribed with advantage, but they should be properly warmed before drinking. *Morphia* (gr. $\frac{1}{12}$–$\frac{1}{8}$ doses),

for the relief of pain. If the epigastrium be sensitive to pressure, then *leeches* or *wet cups* may be applied with advantage.

If there be vomiting, *morphia*, or *ice*, or *ice-water* or *creasote* (gr. iv. to ʒ vi. water in tablespoonful doses), or *tr. iodini*, gr. ij.–iij in sweetened water. Abstinence from all food and rectal alimentation has been highly recommended.

DISEASES OF THE INTESTINES.

CATARRHAL ENTERITIS.

DEFINITION.

Is an inflammation of the intestines, due to hyperæmia, causing excessive transudation of a salty fluid deficient in albumen, or an abnormal production of mucus and cells.

VARIETIES.

Acute and chronic catarrhal enteritis; marasmus; typhlitis; and perityphlitis.

MORBID ANATOMY.

In the acute form.—The changes that take place occur most frequently in the large intestine, about Peyer's patches; less so in the ileum; and rarest in the jejunum and duodenum. After death, Peyer's patches are found to be swollen, as are also the solitary glands, causing them to project above the surface of the mucous membrane. The contents of the intestines are, at first, a serous fluid with detached epithelial and young cells, or a cloudy mucus adherent to the walls of the intestines.

In the chronic form.—The mucous membrane is of a brownish-red, or slate color; puffed up and forming rectal polypoid protrusions. Hypertrophy of the muscular walls often exists, causing constriction. The mucous membrane may, in some cases, appear as if sprinkled with bran (indicating a diphtheritic type.)

There may be ulcerations, either diffused or follicular. The diffuse form of ulcer may be due to foreign bodies, or retention of fæces; as in typhlitis, where the arrest is most probable to take

place. The follicular form occurs exclusively in the large intestine, at the lower part, often causing great destruction of tissue.

ETIOLOGY.

It accompanies obstruction of the liver circulation; disease of the respiratory and circulatory organs, causing obstruction to the vena cava; from disturbance of the external circulation, causing active hyperæmia and catarrh, as caused by burns, or sudden exposure to a low temperature. It occurs in peritonitis, especially the puerperal type, which leads to collateral œdema; from collateral fluxion; from fluxion to the intestinal capillaries induced by mental excitement; from local irritation, as in the case of purgatives; from retention of fæcal matter, causing local peritonitis; from epidemics; and, finally, it accompanies various fevers.

SYMPTOMS.

In the acute form.

Accelerated movement of the intestines.
Diarrhœa—preceded by rumbling; may consist of fæcal matter mixed with cylindrical epithelial cells, or with undigested food. It may be of a greenish color and contain blood.
Pains in the abdomen.
Abdomen—prominent.
Fever—the temperature usually varying between 100°–103°.
Rumbling and gases in the intestines—showing that the disease has extended to the small intestine.
Constant desire to defecate (if the rectum be involved).

In the chronic form.

Secretions are scanty and contain mucus.
Constipation—is usually present.
Debility.
Emaciation.
Complexion—pale, or dirty-gray color.
Abdomen—tense and resistant.
Respiration—impaired.
Congestion of the brain—may occur, resulting in dementia and suicide.
Secretions from the mucous membrane—may be increased.

Peristaltic action—may be increased, (with diarrhœa).

Death may result from exhaustion.

In marasmus.

Occurs from an exhaustive and obstinate diarrhœa (is most common about the end of the first year and after weaning).

Dejections—are thin copious, and watery; clay-colored; fœtid, and mixed with undigested food.

Child becomes flabby and emaciated; fat and muscles are absorbed, and patient looks prematurely aged.

Excoriations about the anus.

Aphthous rash appears in the mouth.

In Typhlitis.

This is due to the muscular coat of the intestine losing its power of contraction.

Fæces are collected in the cæcum and ascending colon.

Constipation and diarrhœa alternately occur.

Passages of mucus or bloody mucous masses are frequently present.

Nausea.

Vomiting.

Pain—in the right inguinal fossa.

Tumor—"sausage shape," and is external to the median line.

Inflammation—which may extend to the serous covering and neighboring organs, causing

Abdominal tenderness—often indicating *Perityphlitis* (for symptoms see page 187).

Follicular ulcers—occur in cachectic persons. At first the symptoms are those of protracted catarrh of the large intestine; but, after a while, there will be found peculiar translucent lumps resembling swelled sago in the mucus, and their passage is preceded by tormina with slight tenesmus.

Adhesion of the liver—producing distortions and contractions of the intestines.

PROGNOSIS.

Is most unfavorable when there are follicular ulcers of the large intestine, particularly if the patient be already cachectic.

TREATMENT.

If it occur *from constipation—leeches* should be applied to the anus.

If *from cold* – send the patient to bed, give him warm drinks of chamomile tea, and apply hot flannels to the abdomen.

If *from a damp climate*—the patient should wear woollen stockings, which should be changed when the feet get cold.

If *from improper nourishment*—the diet should be regulated. The patient should be fed on meat broths, and finely-shaved raw beef, with a little white bread, and a small quantity of good wine. Milk must not be allowed while there is diarrhœa.

If *from retention of fœces*—commence with a purgative and examine the passages.

If the passages be bloody, etc.—give one good large dose of oil and regulate the bowels.

If *from habitual constipation*—administer *pulv. digitalis* and *ext. belladonna*, gr. $\frac{1}{8}$ to $\frac{1}{4}$, in the form of a pill.

If *from typhlitis*—prescribe *ol. ricini*, ʒ ss.-i. If there be no vomiting, an enema, with salt, oil, milk or honey, may be used; and *leeches* and hot poultices applied over the right inguinal region.

If *from cholera morbus*—hot cloths well wrung out should be applied to the abdomen, also *tannin* and *nitrate of silver*, *enema of sulphate of zinc*, tannin, ʒ ss. to ʒ ij. water, or nitrate of silver, gr. ij. to ʒ vi. water. In order *to arrest the diarrhœa*, give mucilaginous drinks, such as rice, oatmeal, or barley water, etc.; the patient should have red wine, infusion of dried whortleberries, and roasted acorns. ℞ Catechu, ʒ ij.; mucilage, ʒ vi.; M. Sig., ʒ i. every two hours; or opium, ↄ j.– ʒ ss. and mucilage ʒ vi.; or a weak solution of ipecac. M. Sig., ʒ i. every hour.

CHOLERA MORBUS.

DEFINITION.

Is that form of acute gastric catarrh which extends to the in-

testinal mucous membrane, and which is characterized by profuse transudation of a fluid into the stomach and intestines.

ETIOLOGY.

Occurs in the summer, and is usually epidemic. It is sometimes excited by error of diet.

SYMPTOMS.

Come on suddenly, usually at night, with a sense of oppression at the pit of the stomach.
Nausea.
Vomiting—often profuse in quantity, and possibly projectile in character.
Borborygmi and abdominal cramps.
Stools—pulpy at first, then thin and liquid.
Intense thirst.
Bloody evacuations—in severe cases.
Urine—diminished in quantity.
Calves of the legs painfully contracted.
If evacuations continue, they become like rice-water discharges.
In children.
Evacuations—are first acid, green or yellowish, then almost white.
Vomiting—but not of curdled milk—resembles bile.
Legs are drawn up.
Convulsions—from anæmia of the brain.

TREATMENT.

For the vomiting and diarrhœa, give *opium;* should they be excessive, give *gr. ss. doses* in the form of a powder. For vomiting, *ice* should be eaten. When there is collapse, *stimulants* should be given inwardly, and hot poultices applied outwardly. For diarrhœa, use *Squibbs' "tinct. opii comp."* When injurious or decomposing food is in the stomach, an *emetic* should be given, and when it has passed into the intestines, *rhubarb* or *compound infusion of senna* should be administered.

PERFORATING DUODENAL ULCER.

MORBID ANATOMY.

The most frequent seat of this disease is in the upper horizontal portion, but sometimes the descending portion of the duodenum is affected. The edges of the ulcer are sharp and not swollen. There is a greater loss of substance in the mucous membrane than the muscular coat. The floor of the ulcer is sometimes formed by neighboring organs, to which the duodenum has become adherent before perforation has taken place. The ulcer may communicate with the gall-bladder and produce an external fistulous opening. Stricture of the duodenum may follow, or obliteration of the ductus communus choledochus.

ETIOLOGY.

This disease is really a necrosis, and a solution of the necrosed part of the intestine by the gastric juice. They occur more frequently in men than in women, while ulcer of the stomach is more frequent in women. It is said to be the particular lesion of burns occurring over the abdomen.

SYMPTOMS.

Are latent until perforation takes place.

Dyspepsia—(slight, and which has remained unnoticed) may have existed.

Pain—may have occurred in the right hypochondrium for several hours after meals.

Perforation may produce sudden death.

TREATMENT.

Strict regulation to the diet should be observed. Alkalies and alkaline mineral waters may be taken freely.

PERITYPHLITIS.

DEFINITION.

Is an inflammation of the connective tissue attaching the cæcum and ascending colon to the iliac fascia.

MORBID ANATOMY.

Abscesses may extend to the region of the kidneys, and, below Poupart's ligament, to the inner part of the thigh. The posterior wall of the cæcum, ascending colon, anterior wall of the abdomen, or skin of the thigh, may be perforated, or the contents of the abscess may escape into the abdominal cavity, and cause peritonitis. The exudation may be absorbed, and recovery ensue, or the inflammation may lead to necrosis of the inflamed connective-tissue.

ETIOLOGY.

It occurs in typhus, septicæmia, puerperal fevers, etc., and may follow typhlitis.

SYMPTOMS.

If from typhlitis, a painful tumor remains lying far back in the abdominal cavity.

Pain—is circumscribed, and a numbness often exists in the right leg, from pressure on the nerve-trunks.

Inability to raise the thigh—because the psoas and iliacus muscles are infiltrated.

Patient lies on the right side, with the body bent forward (so as to relax the psoas and iliacus muscles).

Œdema—from pressure on the veins (sometimes).

Tumor in the abdomen or thigh—which may be fluctuating, if suppuration has taken place.

Ulceration of the vermiform appendix may occur, resulting in symptoms of shock or collapse.

Inflammation of the transverse colon and sigmoid flexure may occur.

Death—may result from exhaustion, or peritonitis.

DIFFERENTIAL DIAGNOSIS.

From typhlitis (see page 183).

TREATMENT.

The abscess may be opened, or the contents evacuated by *aspiration*. If the contents escape into the ascending colon, recovery usually ensues. At the onset of the disease, it is advisable to

apply *leeches* several times, and, so soon as fluctuation takes place, the tumor should be opened.

PERIPROCTITIS.

DEFINITION.

Is an inflammation of the connective-tissue around the rectum, which may develop in the course of an acute and chronic inflammation of the neighboring parts; and as the result of degeneration or traumatism of the rectum.

MORBID ANATOMY.

Acute periproctitis usually leads to abcesses. The chronic form may terminate in thickening and induration of the inflamed connective-tissue, which ends in partial suppuration, forming fistulous openings, which are extremely difficult to heal.

ETIOLOGY.

It accompanies affections of the pelvis or pelvic organs. It is developed sometimes in phthisical patients.

SYMPTOMS.

In the acute form.

Tumor—which is hard and painful. This may be felt in the perineum, or in the cavity of the coccyx.

Infiltration of the connective tissue.

Patient is unable to sit up.

Pain—severe (on defecation).

Tenesmus—severe (in some cases).

Evacuation of purulent matter and formation of fistulæ.

In the chronic form.

Are obscure, till stricture of the rectum is induced.

If abscess form, there is severe pain, with the above-named symptoms superadded.

TREATMENT.

Cold may be applied, or poultices, or fomentations. The tumor should be opened as early as possible to prevent perforation of the bladder or rectum.

DYSENTERY.

SYNONYM.

"Bloody flux."

DEFINITION.

Is an inflammation of the muscular and mucous coats of the large intestines.

VARIETIES.

Acute (which is usually epidemic or sporadic); chronic; typhous; bilious; malarial; ulcerative; "strumous" or tubercular.

MORBID ANATOMY.

There is usually a diphtheritic type of inflammation. The diseased portions of the mucous membrane are infiltrated by a fibrinous exudation, and consequent impairment of nutrition and sloughing follow.

In mild cases.—The mucous membrane of the large intestine is reddened by ecchymotic injections, and infiltrated by a grayish soft exudation which covers the epithelial coat and has the appearance of bran. The submucous tissue is swollen and infiltrated with serum. The serous coat is cloudy and dull (from œdema).

In severe cases.—The mucous membrane is changed to a black friable charred mass, which may be thrown off. The sub-mucous tissue may be infiltrated by a bloody serous fluid, or it may be pale. The superficial blood-vessels are consumed to a black powdered mass, and when this is thrown off, the sub-mucous tissue appears infiltrated with pus. The peritoneal coat loses its lustre and is injected by dilated capillaries covered by a brownish discolored ichorous exudation. The intestine contains an offensive coffee-ground fluid, and is either in a state of passive dilatation, or collapse. The muscular coat is shrunken, pale, faded, and easily torn, and the glands are swollen.

The liver becomes hyperæmic, and abscesses may form within

it. These are to be explained as the result of the following changes: (1) inflammation of the muscular coat of the bowel with ulceration; (2) inflammation of the veins, constituting "phlebitis; (3) blood-clots ("thrombi") form in these veins; (4) suppuration of these thrombi now occurs, causing their disintegration; (5) these disintegrated thrombi are carried, as emboli, to the liver by the portal vein; (6) infarctions of the liver are thus produced, and are followed by suppuration; (7) abscesses in the centre of each infarction, or at the seat of obstruction to any of the portal vessels. This secondary suppuration depends upon the *suppurative origin* of the emboli in the portal vessels.

ETIOLOGY.

Is a disease of hot climates depending on miasm. It occurs in the summer and autumn, and often depends upon some blood-poisons, such as purpura, cholera, fever, ague, syphilis, etc. It may also occur from drinking bad water; from atmospheric changes; from inflammation of the colon; from bad hygiene, exposure, and hardships, as in the army in the field, etc. As in cholera, the stools and water-closets are apt to cause it to spread on account of the germs created therein (Niemeyer).

SYMPTOMS.

In the acute form.

Chill—(which is usually preceded by a diarrhœa).

Fever—slight, with great thirst.

Countenance—anxious and distressed.

Anorexia.

Temperature—elevated.

Pulse—(in severe cases) rapid, full, and marked; (in slight cases) very little excited.

Tongue—furred.

Skin—(in severe cases) is very hot and dry; (in slight cases) cool.

Stools—frequent and semi-feculent; small; bloody, slimy, offensive, and mixed with mucus and pus and cast off epithelium; often "jelly-like," or greenish.

Great *rectal tenesmus* and *purging*.
Irritability of the bladder—(strangury or retention).
Urine—high-colored and scanty.
Irritation of the vagina—(in female patients).
Pain—griping in character and associated with tenderness on pressure (about the colon).
Constipation, (alternating with diarrhœa) which may be permanent if stricture of the intestine be produced.
Nausea and vomiting.
Collapse.

In the chronic form.

Is associated with a chronic diarrhœa.
Rectal tenesmus—(often marked and distressing).
Stools—same as in the acute form, and also loose and frequent.
Discharges—alvine.
Pain—griping.
Emaciation.
Extensive inflammation of the mucous membrane of the colon.
Examination of the rectum shows *ulcers*.
Paralysis of the sphincter muscles may occur.
Death—may result from paralysis of the intestines.

PROGNOSIS.

In the acute form.—The patient may die (if an epidemic exists) in a short time.

In the chronic form.—The prospect of complete recovery is doubtful.

TREATMENT.

In the acute form.—The patient should be kept in bed, and fed on milk, arrow-root, broth, and the whites of raw eggs, etc. *Tincture opii* should be given in full doses; or *oleum ricini et opii* in small doses.

In the chronic form.—The patient should be put to bed and kept perfectly quiet; he should be fed in the same manner as in the acute form. Full doses of *tinct. opii* should be given to relieve

the pain, and *oleum ricini et opii* in small doses when the dejections contain no fæcal matter for a day or two. Should the discharges be excessive and bloody in character, injections of starch and laudanum, or starch and iodoform, or a suppository of *morph.* gr. $\frac{1}{8}$, *ext. belladonna* gr. ss. will frequently arrest this symptom in a very few hours. *Ipecac* (gr. xxx. every half-hour till retained) is highly recommended by eminent physicians in the East Indies, where this disease is more prevalent than in any other part of the world; and remarkable results seem to have been obtained by this treatment.

ASIATIC CHOLERA.

MORBID ANATOMY.

There is, at first, terrible emaciation. The skin is corrugated. Black circles will be seen around the orbit, and flattening of the eye, from absorption of its fluids. Dryness of the muscles exists (from absorption of fluid), and contraction of the muscles after death often occurs. Elevation of the temperature takes place after death, and the vessels are found to be filled with coagulated blood.

On opening the body, the right heart will be found filled with blood; the organs very dry; and the alimentary canal injected and filled with the so-called "rice-water" discharges.

STAGES.

1st. Painless diarrhœa.
2d. Rice-water discharges and cramps.
3d. Collapse.

ETIOLOGY.

The rice germ is claimed to be the cause of this disease; and the discharges of the patient are supposed to be contagious. Mental influence (fear, etc.) may predispose to it.

SYMPTOMS.

1st stage.—Painless and feculent diarrhœa.
2d stage.—Profuse rice-watery discharges from the bowels.

Projectile vomiting of rice-watery discharges (*this is characteristic*).

Terrific cramps.

Insatiable thirst, (due to abstraction of water from blood.)

Terrible emaciation (from extraction of water from the tissues).

Premature aging of the patient (from the emaciation and abstraction of fluids from the tissues).

Stoppage of pulse in the extremities (from thickening of the blood due to abstraction of its water).

High temperature.

Collapse—the symptoms of which are:

Complete suppression of urine.

Cessation of the "rice-water" discharges.

Insensibility.

Fæces passed involuntarily and in small quantities.

Marked cyanosis.

3*d stage.*—Coma and death.

Duration.—From the time the rice-watery discharges commence, 24 to 72 hours may elapse before death occurs.

TREATMENT.

Stop the diarrhœa, in the first stage, by *rest in bed;* give *large doses of opium* and remove the patient from all influences favoring the disease.

CARCINOMA OF THE INTESTINES.

MORBID ANATOMY.

It affects the large intestines, especially at the sigmoid flexure and rectum; it may also occur in the small intestines.

The scirrhus form attacks the sub-mucous connective-tissue, and, when it has perforated the mucous membrane, medullary masses arise from the scirrhus base, and form a ring-like structure. The cancer may extend to the peritoneum, the mesentery, or neighboring organs. The intestines may become adherent to the abdominal walls. Fæcal fistula, or perforation of the vagina

or bladder may occur through breaking down of the cancer (if situated in the rectum).

ETIOLOGY.

Is obscure. May be due to hereditary predisposition.

SYMPTOMS.

Pain—sometimes sharp and lancinating, but usually dull and confined to one point.

Tumor—uneven, nodular, hard, and painful; at first it may be movable.

Habitual constipation, (from degeneration of muscular coat).

Rapid loss of strength and emaciation.

Cachexia.

If it occur in the rectum.

Pain - severe about the sacrum, extending to the back and thighs.

Dilatation of the hemorrhoidal veins.

Passage of bloody mucous.

Habitual constipation or profuse diarrhœa, which cannot be stopped.

Diminution of the rectal calibre (on a digital examination).

TREATMENT.

Is palliative. *Castor oil* may be given daily to evacuate the bowels. The food should consist of concentrated broths, soft boiled eggs, and milk. If confined to the rectum, surgical interference may be demanded.

INTESTINAL HEMORRHAGE.

MORBID ANATOMY.

The mucous membrane is suffused with blood, after capillary hemorrhage takes place. The blood is usually of a chocolate-brown color, or it consists of a black, tarry mass. Hemorrhoids frequently occur, which are at first bluish in color, and with thin

walls; if they continue for any length of time, and become chronic, they lose that bluish look, become hard, since their walls become thickened.

ETIOLOGY.

From cirrhosis of the liver; erosion of vessels from ulcerations (occurring in typhoid fever, dysentery, and tuberculosis of the intestines); disease of the walls of the vessels; from escape of blood from hemorrhoids, caused by collection of fæces in the rectum, producing rupture of the hemorrhoids.

SYMPTOMS.

Blood is frequently passed in small quantities, with the discharges of the disease with which it occurs.

Hemorrhoids.—When this complication occurs, there will be pain when the stools are hard (if the varices are small); the pain may be constant and the patient unable to sit down (due often to pressure of the sphincter muscle of the rectum upon them).

Bleeding—occurs during defecation.

TREATMENT.

Acetate of lead, with opium, may be given either by the mouth or rectum; *tannic* or *gallic acid* in the form of a pill or solution; in this latter it is used as an injection; or any of the astringents may be resorted to. Should the patient suffer greatly from hemorrhoids, the diet should be regulated. Meat and eggs should only be taken once a day, and then only in small quantities; the principal diet should consist of vegetables, fruit, rice, etc. *Stimulants must be forbidden.* The patient should drink freely of water; take long walks, and resort to energetic muscular exercise.

INTESTINAL OBSTRUCTION.

MORBID ANATOMY.

Above the constriction, the intestines are dilated and elongated, and the walls hypertrophied and thickened. The vessels are

compressed, and great capillary congestion, and probably stasis, occurs, and, as a result, mortification of the part occasionally ensues. Below the constriction, the intestines are empty and collapsed.

ETIOLOGY.

From compression, resulting in contraction and closure, where the rectum is compressed by a retroverted uterus, tumors, abscess of the pelvic bones, or an overfilled portion of the intestine; from structural changes causing constriction, resulting from cicatrization of intestinal ulcers (catarrhal, follicular, dysenteric), or syphilitic ulcers of the rectum, or carcinoma; from closure produced by volvulus, or from entanglement of the mesentery with the intestine; from internal strangulation, or incarceration; from bands resulting from peritonitis, especially between the uterus and its surroundings; from invagination or intussusception, occurring both in the large and small intestine; from sedentary habits; from impaction of fæces: from hernia; from passage of gall-stones; from rents in the mesentery, etc.

SYMPTOMS.

If partial closure only exist.

Defecation—tedious and difficult.
Feet—may be very cold (from impeded return of blood from the feet) due to habitual constipation.
Habitual constipation.
Menstruation—abundant (in women).
Frequent erections and sexual emissions (in males).
Neuralgic pains in the legs—(from overladen rectum).
Small fæces—(like those of sheep, etc.) in case of stricture.
Prominence of the abdomen (from tympanites).

If complete closure exist.

Face—distorted and pale and ghastly.
Hands—cold.
Pulse—small and imperceptible.
Prominence of the abdomen—(from typanites).
Jactitations, or cramps.
Obstinate constipation unrelieved by treatment.

Vomiting—feculent in character.
Death—may result from general paralysis.

If closure exist with peritonitis.

Abdomen—puffed up and extremely tense.
Great pain—on the slightest pressure.
All movements of the body are avoided.
Temperature—high.
Pulse—frequent, hard and tense.
Respiration—hastened and shallow (from compression of the lungs by tympanites, and from the pain).
Cyanotic look.
Perforation — especially if the disease occur suddenly, accompanied by sudden pain and symptoms of shock or collapse.

If intussusception occur.

Sausage-shaped tumor (felt on palpation of abdomen in the right inguinal region).
There may be bloody or mucous passages.

TREATMENT.

For constriction.—The diet should consist of eggs, meat, strong broths (muscular meat with delicate fibre), and evacuation of the bowels by *laxatives* or *enema.*

For invagination.—Gastrotomy should be resorted to.

WORMS.

VARIETIES.

Tænia solium—(long "tape" or "chain-worm") are usually found in the small intestine, but may occur in the large intestine.

Tænia mediocanellata—are very like the tænia solium.

Bothriocephalus latus –broad tape-worm, which closely resembles the tænia solium, and is also found in the small intestine.

Ascaris lumbricoides—round worm—found in the large and small intestine, and may enter the œsophagus, larynx, gall-duct, or ductus communis choledochus.

Oxyuris vermicularis—("thread-worm")—found usually in the rectum. They may enter the vagina from the anus, and be found in that canal.

Trichocephalus dispar—("whip-worm")—found in the large intestine, especially in the cæcum.

SYMPTOMS.

In some cases the patient is in no way affected (especially in the case of tape-worms), while others may have any of the following symptoms:

Severe abdominal pain—(as twistings and turnings).
Nausea.
Vomiting.
Emaciation. Salivation in some cases.
Diarrhœa—and occasional passage of worms.
Dilatation of the pupil.
Epilepsy. } most common in children.
Chorea.
Intestinal obstruction (from the round-worm).
Pruritus ani (especially late in the evening and during the night).
Incessant desire to defecate.

TREATMENT.

Pork must be strictly prohibited.

For tape-worm.—Filix mas ʒ ss.–i. t. d. s. It should be taken the first thing in the morning after fasting (the fasting must be continued during the whole of the day), and the last dose to be taken at bed time. Next morning, after the patient has fasted twenty-four hours, a good laxative of *calomel*, *scammony*, or ʒ i. *ol. ricini* should be administered. *Pomegranate-rind*, ℥ iv. to O. i.–ij. water, and macerated for twenty-four hours, may be used. This should be boiled till reduced to one-half of the quantity; then it should be divided into three doses, and taken while fasting for twenty-four hours. Should the worms not have passed after having taken the third dose, give *ol. ricini*, ʒ i.–ij.; or, *oleum terebinthi*, ʒ i.–ij. mixed with honey; or, *ol. ricini* at bed-time will usually have the effect of removing them.

For "round-worms"—Troches of santonin, ea. gr. ss.-i.

For "thread-worms"—large enemata of cold water and vinegar, or *corrosive sublimate*, gr. $\frac{1}{4}$–$\frac{\text{ʒ}}{3}$ ij. of water (used as an enema) have been advocated.

Prior to administering any of the above remedies, the patient should live moderately; his bowels should be kept open; and should be made to live on herrings, ham, onions, and salty or spicy food. Strawberries, huckleberries, or blackberries may be eaten freely.

INTESTINAL COLIC.

SYNONYM.

"Enteralgia."

DEFINITION.

Is a term applied to all painful affections of the intestines, which are not caused by inflammation, or textural changes of the intestinal walls, but by irritation of the peripheral extremities of the intestinal nerves.

VARIETIES.

Those which are most common are *lead*, *flatulent*, and *bilious colic*.

ETIOLOGY.

It occurs in the middle and lower part of the abdomen, from structural disease of the ganglia and plexus of the sympathetic nerves; from mesenteric neuralgia in females; from worms: from eating unripe fruit; from food which is difficult of digestion; from accumulation of fæces; hardened fæces; from obstinate constipation; from fright; from anger; from exposure to cold; from affections of various organs, as a morbid state of the liver, kidneys, bladder, testicle, uterus, ovaries, etc.; from an abnormal state of the blood.

Lead colic—occurs from poisoning by drinking water carried through leaden pipes, producing disturbance of the motor (hyperkinesis), as well as of the sensory nerves (hyperæsthesia); also in painters, and those employed in mixing colors.

Flatulent colic—occurs from distention of the intestines with gas.

SYMPTOMS.

Pain—from the navel extending over the abdomen; is of a tearing, cutting, pressing, twisting, pinching, or bearing-down character. Is usually relieved by firm pressure.
Jactitations—patient seeks relief by compressing the abdomen.
Hands, feet, and cheeks are cold.
Features—pinched.
Countenance—agonized and distorted.
Pulse—small and hard.
Nausea.
Vomiting.
Desire to defecate.
Tenesmus.
Constipation.

Lead colic.

Symptoms are generally preceded by those of lead poisoning.
Skin—looks dirty.
Pain—severe (extending to the back and extremities).
Blue line along the gums and bad breath.
Sweetish taste in the mouth.
Pulse—slow.
Voice—occasionally lost (aphonia).
Constipation—obstinate (from constriction).
Abdominal walls strongly contracted, and the abdomen as hard as a board.

Flatulent colic.

Pain—may be very severe, but relieved by pressure.
Countenance—expression of anxiety.
Body—covered with a cold sweat.
Face—pale and distorted.
Pulse—small.
Nausea. } present in some subjects.
Vomiting. }
Strangury. }

TREATMENT.

Opium should be given in all forms; it acts as an anæsthetic.

For lead colic.—*Sulphuric acids* and *sulphates, Glauber's salts*; *iodide of potash*, gr. xv., frequent bathing, and change of clothing. The patient, if a painter, should be forbidden to continue in the business.

For flatulent colic.—Warm teas should be given, made of either *chamomile*, *valerian*, or *peppermint;* they should be drank quickly and very hot. *Enemas* should be resorted to; also flannels wrung out in hot water and sprinkled with turpentine, and applied over the abdomen.

For foreign bodies.—*Ol. ricini*, or an enema, should be given; or warm compresses may be applied over the abdomen.

PERITONITIS.

Is an inflammation of the peritoneum. The peritoneum is the largest serous membrane in the body; its office is to facilitate the motion of the intestines. Many gallons of fluid may be contained in the peritoneum; as many as twenty quarts are often removed. Predisposition to peritonitis may occur from any abnormal position of the viscera.

VARIETIES.

1. *Acute primary peritonitis*—which may be due to infection.

2. *Infectious primary peritonitis*—which depends upon no local cause, except blood changes, which cause its development, as in puerperal and scarlet fever, or general blood-infection from severer poisons.

3. *Chronic peritonitis.*—This form simply differs from the acute, in that it is slower of development, and associated with some other cause, as cancer, tubercle, and various new growths.

ACUTE PRIMARY PERITONITIS.

MORBID ANATOMY.

The same changes take place in this as in all serous cavities which consist of (1) reddening of the surface—from changes in

the serous membrane and sub serous-tissue—(2) loss of lustre of the membrane—(3) shedding of epithelium and an infiltration of the epithelial cells—(4) pouring out of a plastic exudation, and, underneath this, a layer of embryonic cells—(5) a serous exudation takes place, which washes off the plastic exudation—(6) the formation of new connective-tissue which contains new blood-vessels with thin walls—(7) adhesions of the two surfaces, from the formation of new connective-tissue. When these changes take place slowly, there will be found under the plastic layer projecting prominences of connective-tissue which may touch each other, causing adhesions between the walls of the abdominal cavity and those covering the intestines, or, the bands may strike across the intestines, causing contraction. A loop of intestines may sometimes get caught in this stricture, producing internal hernia, etc., when it will be impossible to remove the adhesions without lacerating or tearing the intestines. When purulent exudation occurs, and it is abundant, it may undergo absorption, or become encapsulated. Calcareous exudation may take place in the cavity. In addition to the adhesions and bridles, there may be thickenings of the peritoneum, which, in places, may become from one-quarter to one-half inch thick. Adhesions may form over an organ, causing contraction, and such pressure on some of the organs may produce disordered function. These latter changes are most important when occurring in the uterus, since, after a while, they may result in constriction of the ovaries and Fallopian tubes.

ETIOLOGY.

From intestinal obstruction, perforation, and ulceration; typhlitis and perityphlitis; perforation of the intestines and stomach, from any cause; perforation of the intestines in typhoid fever; abscess of the liver; perforation of the diaphragm in emphysema; rupture of the bladder or gall-bladder; from strangulated hernia; inflammation of the muscular coat of the intestines or stomach; rupture of an aneurism into the peritoneum; peri-nephritic abscess; and, finally, *the most common is a change in the vermiform appendix, as ulceration and escape of gas into the peritoneal cavity. Such escape of gas into the peritoneal cavity from per-*

foration is always fatal, and that in a very few hours. In addition to the causes above-named, inflammation of the liver; cancerous development of the stomach, from pressure on the peritoneum (localized peritonitis); inflammation of the uterus, or ovaries, etc.; infectious causes, as pyæmia, septicæmia, puerperal fever; and the abuse of alcohol (when you may have a general peritonitis) may prove exciting causes of this disease.

SYMPTOMS.

In the early stage.

The ordinary duration is from two to eight days—rarely over a week.

Pain—in the right inguinal and umbilical regions, which, for a few hours, can be covered with the palm of the hand; it gradually extends, in about three or four hours, over the entire abdomen. It is increased on firm pressure, especially in the region where it commenced (*pressure should be made with the palm of the hand*, and the countenance of the patient watched).

Countenance—pale, anxious, and bears an expression of anguish.

Pulse—accelerated; and, at the end of twelve hours, rises to 100; is, usually, firm and incompressible.

Abdomen—distended (after twelve hours), especially in the region of the pain.

Legs—*flexed on the abdomen*—the patient keeps a position on his back, and does not move from it.

Respiration—no abdominal movement; only thoracic breathing is perceptible (as the abdominal pain prevents motion).

Fear of pain on attempt to move patient.

Tympanites—(due to accumulation of gas, because the inflammation has arrested the peristaltic action of the intestines).

Vomiting—at the end of twenty-four hours. The vomited material is regurgitant in character, and the expulsive effort increases the pain; there is at first a discharge of the contents of the stomach, after which it assumes *the*

characteristic spinach-green vomiting; it may be stercoraceous in character. This is an important sign, as this does not come on till late; but, should it come on early, it indicates that there is extensive intestinal obstruction. It may be *coffee-ground* in appearance (from capillary hemorrhage of the stomach), when there is inflammation around the stomach obstructing the circulation (this may be due to infectious causes).

Hiccough—may be present, due to the involving of that portion of the peritoneum covering the diaphragm.

At the end of the second day.

Tympanites—(from the same cause as above) is increased.

Pulse—accelerated 120 (usually small and thready).

Countenance—more anxious.

Cyanosis.

Respiration—hurried and thoracic; may be short and catching (due to tympanites).

Constipation—is a constant attendant of the disease.

By the fourth or fifth day.

Patient lies motionless on the back.

Pulse—very feeble.

Respiration—short and shallow.

Mind—clear up to the last; and, before death, is clearer than in any other disease.

Cyanosis—extreme.

Perspiration—profuse.

Collapse—(pulse is imperceptible and respiration more shallow).

Death—is quiet and peaceful.

In case of perforation, the disease may not last more than twenty-four hours, and is accompanied by the following symptoms:

Shock—causing collapse.

Symptoms are rapidly developed.

Countenance—bears an anxious expression.

Pulse—almost imperceptible.

Abdomen—suddenly distended.

Coldness—suddenly developed.

Cyanosis—suddenly developed.
Vomiting—suddenly developed.
Respiration—thoracic and shallow.
Death—due to the effects of the shock on the nervous system. An apparent reaction may take place, in some cases, and life be prolonged for two or three days.

From the discharge of pus into the peritoneal cavity.

The symptoms are rapid, but not so sudden or rapid as in perforation.

DIFFERENTIAL DIAGNOSIS.

It may be mistaken for enteritis; colic; passage of renal, or bilious calculi; rheumatism; hysteria; or lead poisoning.

PROGNOSIS.

Depends greatly upon the variety. *If localized*, and seen early, the prognosis is good; if from *ulceration*, which excites peritonitis, a moderately good prognosis may be given; if from *typhlitis* and *perityphlitis*, the prognosis is good, if there be no ulceration, otherwise it is bad. *Perforation of the intestines and entrance of gas into the peritoneum is fatal in a few hours.* In cases of *traumatism*, where the intestines escape injury, the prognosis is good. So long as the disease is local in character, and not infectious, and no perforation exists, the prognosis is good; but, should it become general, even after it has been localized for some time, the prognosis is grave.

TREATMENT.

The *opium plan* of treatment is the only way of handling this disease; in may be *given in gr. i.–v. doses*, or *hypodermically* ♏ *xx. of Magendie's solution* every hour till the respirations reach *twelve per minute.* Opium acts as a controlling influence on peristaltic movement, also as an antiphlogistic, and it controls the inflammatory conditions. What are the effects of opium, and what are the doses that should be administered? So soon as you obtain the proper effect of the drug, you will observe the following symptoms in the patient: (1) contracted pupils; (2) suffusion of the eyes; (3) dull countenance; (4) profuse perspiration; (5) rash on the nose,

which extends down the neck, and is accompanied by intense itching; (6) the respiration should grow less and less until twelve a minute has been reached, *when great caution must be observed*, as the patient is semi-narcotized, and it should always be borne in mind that *the slowness of the respiration is the respiration of the drug;* (7) the pulse is less frequent, having reached 90 in a minute, and full and strong, which, previous to the treatment, was hard and 110 in a minute; (8) entire abolition of pain; (9) subsidence of tympanites; *this is the proof of the cure*, and the treatment should gradually be stopped.

It is not the amount of sleep you have to fear, as the patient may be so affected that from the very onset, but it is *the profoundness of the sleep;* for, if you cannot rouse him or make him swallow, the treatment should be discontinued, and that before he shall have reached this condition. If the respirations should ever drop to *four a minute*, then stimulants should be given, and you must *wait and watch;* but, in such a case as this, *the heart is your compass*, for you will find it beating steadily at 90, and you are safe. Wait until the respiration has reached fifteen to twenty a minute before commencing again, but still you must watch the patient, and *with the utmost care.* The bowels will be constipated, on account of the peristaltic quietening effect of the drug you are using, and, despite the fact that the old women will tell you to move the bowels, *see that they are not moved for two weeks.* After that time, should you deem it advisable to have them moved, use the following enema: Warm water, O. i., and glycerin, ℥ i.-ij., rather than oleum ricini. *Never use cathartics.*

Turpentine stupes have been suggested and used over the abdomen. Leeches may be applied over the point where the disease starts, and followed by an ice-bag or heat; *but there is nothing like opium and allowing the patient to remain in peace.*

INFECTIOUS PERITONITIS.

DEFINITION.

Is that form of peritonitis which depends upon no local cause

except the blood-changes which tend to favor its development, as in puerperal and scarlet fevers, or general blood-infection from some severe type of poison.

SYMPTOMS.

Chill—long and continued.
Temperature—high (even before the chill ceases).
Pulse—120 to 140 (within the first twenty-four hours), and is frequent.
Pain—is first localized, but soon becomes general.
Expression—anxious.
Abdomen—greatly distended.
Vomiting.
Diarrhœa—which is followed by constipation.
Bladder becomes involved.
Delirium.
Cyanosis—after two or three days.
Death.

PROGNOSIS.

Is bad.

TREATMENT.

In consequence of the symptoms of this disease being so sudden in their nature, opium is not sufficient to control the inflammatory process that takes place. Furthermore, the depression of the vital powers is so great that the patient cannot be brought properly under its effect.

It will be necessary to reduce the temperature and pulse, and also to sustain the vitality of the patient until he has withstood the shock of the pus formation and peritonitis. In order to accomplish this end, *quinine* should be given in antipyretic dose; and, in the event that this drug does not have the desired effect, in from twenty-four to forty-eight hours, salicine should be administered in its place. In the event of this disease occurring from puerperal fever, the *saturated tincture of veratrum viride* (gtt. iv.–v.) with *opium* should be given in order to allay pain. This should be pushed (by giving one to two drops of ver. viride every

two hours), as, in a few hours or two days at the most, the veratrum viride will lose its power over the heart. Fear of poisoning need not be entertained *until* vomiting takes place, when it will be necessary to stop the treatment. *Stimulants* should be administered early and freely in large quantities. These are the only four weapons that you will require in attempts to overcome so formidable a disease.

CHRONIC PERITONITIS.

MORBID ANATOMY.

Thickenings of the surfaces of the peritoneum take place, which are interstitial in character. These thickenings may be one-half an inch in thickness, resulting from connective-tissue formation. These thickenings are usually associated with cancer and tubercle. As regards the tubercle, it is the result of a tubercular inflammation which may extend over the whole of the internal, or partially over the external surface, resulting in the omentum being full of these deposits. Adhesions of the two surfaces and binding of the intestines may occur. The gut may be distended or diminished in calibre. There may be a sero-purulent exudation in the peritoneal cavity, or the effusion may fill the whole of the peritoneal sac and only a slight quantity of pus exist. Should the peritonitis be local in character, it affects the organs in the vicinity; as, for instance, in the region of the liver, it will affect the capsule of Glisson and produce hepatitis, or the peritonitis may extend upward from the pelvis to the liver, and involve each organ in its course separately.

ETIOLOGY.

It is usually a sequela of the acute form, a low grade of inflammation, which has been left behind after an acute attack, producing interstitial changes. It may also occur from tubercular or cancerous development, and from traumatism.

SYMPTOMS.

Obscure (if it does not follow the acute form).

Pain—if it follow the acute form—which has lasted a long time; is increased on motion; and the patient may be unable to take exercise.
Loss of strength.
Emaciation.
Digestion—impaired.
Mesenteric glands—enlarged.
Tumor—(localized, along the line of the intestines).
Fluid in the peritoneal cavity—(occurring late in the disease).
If *tubercular in character.*
Pain—in the abdomen.
Digestion—disturbed.
Diarrhœa—(usually resulting in constipation).
Abdomen—distended.
Fever and sweating in the early morning hours.
Impairment usually of some function; thus the urine may be disturbed, or even the stomach may suffer, if the peritonitis shall have extended so high up.

PROGNOSIS.

If it be *general* in character, the prognosis is bad; but should it be *local*, the prognosis will depend upon the amount of constriction. Complete recovery is rarely reached.

TREATMENT.

For the general form—(as it is usually accompanied by fluid in the peritoneal cavity, with rapid emaciation and cachexia) you will only be able to alleviate the symptoms. In order to do this, *oleum morrhuæ* and *iron* and *tonics* should be given, and the nutrition increased.

For the local form—(provided it be not due to tubercle or cancer)—a permanent counter-irritation should be established by a seton or continuous blister, and the nutrition carried to the highest possible point. Should there be a large amount of fluid in the peritoneal cavity, it must be removed with great care.

DISEASES OF THE LIVER.

CIRRHOSIS OF THE LIVER.

DEFINITION.

Is a condition characterized by excessive connective-tissue development.

STAGES.

1*st.* *Enlargement*—dependent upon hyperæmia and cell-proliferation of connective tissue.

2*d.* *Organization of this cell-growth into new connective-tissue.*

3*d.* *Contraction of the new connective-tissue*—producing *atrophy* of the liver-tissue from degeneration (on account of pressure on the blood-vessels).

MORBID ANATOMY.

In the first stage—the liver is uniformly enlarged, firm, tough, of a brownish-yellow color, with a smooth surface and rounded edges. The connective-tissue is increased throughout the organ.

In the second and third stages—the liver is small, of a whitish color, the surface is puckered, and the edges are sharp.

On section—there will be great increase in the connective-tissue, the liver substance will appear insular, and of a deep-yellow color.

ETIOLOGY.

Alcohol on an empty stomach. The alcohol is absorbed by the stomach and taken to the liver, causing irritation of its tissues, producing cell-growth. Syphilis; hereditary tendency to cancer or gout, etc.

SYMPTOMS.

In the first stage.

Pain—on pressure, under the ribs.

Sense of fullness and uneasiness in the abdomen.

The liver is found to be enlarged (due to cell growth).

In the second stage.

Liver—(increased in size and smooth in the first stage), after which there will be shrinking of the liver, and

Portal obstruction, as shown by the following symptoms:

In the inferior mesenteric vein—producing hemorrhoids.

In the superior mesenteric vein — producing intestinal catarrh (due to hyperæmia).

In the splenic vein—producing enlarged spleen.

In the gastric vein—producing gastric catarrh, gastric hemorrhage, rupture, and intestinal hemorrhage.

From pressure on the radicles of the portal system—producing jaundice, ascites, dropsy, caput Medusæ, large superficial veins (an evidence of an attempt to establish collateral circulation between systemic and portal systems of veins).

Collateral portal circulation (in case of such obstruction).

1st. Between the superior and the middle hemorrhoidal veins.

2d. Between new vessels formed by adhesions between the liver and diaphragm, or, between the liver and the abdominal walls.

3d. Between the phrenic veins and some vessels on the surface of the liver.

4th. Between the renal and portal veins.

5th. By a dilatation of the umbilical vein, thus joining the abdominal veins at the navel (caput Medusæ).

Palpation—the liver will be decreased in size, hob-nailed, and firm.

Percussion — diminished area of liver dullness; spleen—enlarged.

In later stages.

Ascites—as shown by enlarged abdomen; fluctuation; abdomen becomes flat when the patient is lying down; absence of aortic pulsation; absence of any local tumor; tympanites in front, which changes with the position of the patient; œdema of the limbs (from pressure on veins of pelvis and abdomen). Jaundice—Intestinal catarrh—Fæces, clay-colored—Anasarca may be detected.

CAUSES OF DEATH.

Death may occur from dyspnœa; repeated hemorrhages from the stomach or intestinal canal; intercurrent diseases, or exhaustion; pulmonary œdema.

DIFFERENTIAL DIAGNOSIS.

Between cancer or tuberculosis of the peritoneum. In first stage, from waxy or fatty liver, or other enlargements of the organ.

TREATMENT.

In the first stage.—Strictly forbid the use of liquors. *Leeches* may be applied at the anus (so as to affect the *portal system*). *Saline cathartics* may be administered, such as mineral waters, and alkaline carbonates (potash and soda), or, if the nutrition has suffered, give *iron* (in the form of iron springs).

In the second stage.—Alkaline carbonates should be given to overcome the gastric and intestinal catarrh (they decrease the toughness of the mucus, and enable the mucous membrane to get rid of its mucous coating more readily). *Tapping* should be resorted to for ascites if necessary (always compress the abdomen during the operation and afterward by bandages). Improve the strength and nutrition of the patient by nutritious diet and preparations of *iron*. The diet should consist of milk and eggs.

ASCITES.

The accumulation of fluid in the abdomen may be produced by changes which take place in any of the following regions: the liver, heart, lungs, kidney, and peritoneum. It may also be due to blood-causes.

MORBID ANATOMY.

The amount of serum in the abdominal cavity varies from a few pounds to forty, or even more. The fluid may be clear or cloudy (due to cast off and fatty epithelium). It is usually of a bright yellow color, and rarely contains flocculi of coagulated fibrin.

ETIOLOGY.

See page 26.

SYMPTOMS.

RATIONAL.

Feeling of fullness in the abdomen (becoming painful to pressure).
Slight difficulty on deep inspiration (gradually increasing to dyspnœa, as the abdomen enlarges).
Constipation—from pressure of fluid on the rectum.
Urine—diminished.
Obstruction to the circulation of the lower extremities (from pressure on the inferior vena cava and iliac veins).
Superficial gangrene—may occur if the venous return be greatly embarrassed by the pressnre of the accumulated fluid.

PHYSICAL.

Inspection.—Distention of the abdomen, and a change of its shape with position of the patient (due to the amount of fluid present).
Percussion.—General fluctuation throughout the abdomen.
Tympanites in front, when the patient lies on the back (on account of intestines floating upon the fluid).
Flatness or dullness at the sides (when the patient lies on the back).
Displaced cervix (perceived by digital examination) in the female.

DIFFERENTIAL DIAGNOSIS.

From ovarian dropsy.—(Bamberger says between the crest of the ilium and the 12th rib you find the sound of the large intestine, which you do not observe in ascites.) From cancer; from tuberculosis of other organs (the urine will decide this point, as in tuberculosis it is normal, while in ascites the urine contains traces of coloring matter of bile or abnormal pigment).

TREATMENT.

Tapping should only be performed where life is immediately endangered by obstruction of respiration or threatened gangrene of the skin. *Heim's pills* will be found advantageous in these cases.

JAUNDICE.

VARIETIES.

Hepatogenous—where there is obstruction to the bile-ducts.

Hematogenous, or "non-obstructive," where alteration occurs in the blood-pigments, causing effects similar to those of bile when absorbed.

ETIOLOGY.

See page 25.

SYMPTOMS.

Hepatogenous variety.

Liver and gall-bladder—enlarged.

Urine—peculiar color (light-brown, like thin beer, or dark, like porter). After exposure to the air it becomes greenish, and answers to tests for bile.

Fæces—light, or clay color, or complete decoloration.

Skin—golden-yellow (sometimes only slightly tinged); afterward becoming greenish, or even mahogany color.

Sclerotic of the eye—yellow.

Emaciation—rapid.

Languor and sleepiness.

Pulse—slow (due to the presence of bile acids in the blood).

Itching of the skin—sometimes intense.

Cerebral symptoms—(paralysis, delirium, coma, etc.)

Hematogenous variety.

Fæces—may be normal, or very dark color.

Heart-beat and pulse—irregular and intermittent.

Urine—discoloration is very slight in proportion to the color of the skin, and contains albumen.

Disturbance of the nervous system.

CAUSES OF DEATH.

May occur from intestinal hemorrhage; gastric hemorrhage; dropsy; or marasmus (senile).

TREATMENT.

Improve the state of the patient by proper diet, such as cold meat, strong soup (neither fats, butter, nor gravies should be allowed). *Slight drastics* may be administered (such as *inf. sennæ, rhei, et aloes*) to keep the bowels open. When the urine is diminished, *bitartrate of potash*, *cream of tartar*, or *acetate and carbonate of potash* may be given.

FATTY LIVER.

VARIETIES.

1. From superfluous fat deposited in the liver-cells (Frerichs' fatty liver).

2. From disturbed nutrition of the liver-cells by diseased parenchyma (the cells undergoing a retrograde metamorphosis).

MORBID ANATOMY.

In some cases the liver will be found enlarged and appear flattened, and its edges thickened and rounded; while in others there may be only a slight alteration. The peritoneal covering is transparent, smooth, glistening, and may be traversed by varicose vessels. The liver surface may be yellowish-red or yellow; it feels doughy and pits on pressure. On section, a coating of fat remains on the knife-blade. On *microscopical examination*, the liver lobules may appear filled with fat globules, or individual cells, may be distended by one large drop of oily matter. *This infiltration begins at the periphery of the lobules* near the interlobular veins rarely extending to the vicinity of the central vein.

ETIOLOGY.

From excessive nourishment and no exercise, thus generating too much hydrocarbons; from spirituous liquor (retarding the transformation of tissue); from tuberculosis of the lungs; from incomplete oxidation of hydrocarbons and transformation into fat; from tuberculosis of the bones; from tuberculosis of the intestines; from obesity. (Budd and Frerichs say it may be due to too much fat in the blood, or, when occurring with tuberculosis of the lungs, from giving so much oleum morrhuæ.)—Yellow Atrophy.

SYMPTOMS.

Rational.

Extreme debility.
Gastric and intestinal catarrh.
Diarrhœa.

PHYSICAL.

Inspection.—Fullness of abdomen.

Palpation.—The liver is enlarged and elongated, relaxed and painless, and has thick edges. The liver of drunkards is thus enlarged, and there will be fullness in the right hypochondrium.

TREATMENT.

The patient should be made to have proper hours for exercise. No afternoon naps or liquors should be allowed. The meals must be regular (no gravies, fats, butter, etc.). Karlsbad or Vichy mineral waters, alkaline saline springs, or plenty of soda water should be freely indulged in.

CANCER OF THE LIVER.

Is principally of the medullary type. It may be *circumscribed* or sharply bounded; or *diffused* between the liver-cells, thus showing no sharp line of demarcation.

MORBID ANATOMY.

In the circumscribed variety—the tumor is rounded or globular, and lobulated; is inclosed in a delicate vascular connective-tissue capsule, and, where it touches the peritoneum, is often flattened or has a shallow excavation, called "cancer navel." The tumors may be solitary or innumerable, and may be from the size of a pea to that of a child's head, thus giving the hob-nailed feel to the liver. They resemble brain substance, and contain a large amount of "cancer juice." They may be milk-white or reddish (on account of the number of vessels they contain), or dark-red (from effusion of blood), or black (from the amount of pigment contained). The unaffected portion of the liver is hyperæmic (causing increased size of the liver), and is usually of a yellow color, from compression of the gall-ducts and retention of bile. The liver-cells will be found to have undergone fatty degeneration. There may be adhesions and thickenings with neighboring parts (from chronic local peritonitis). The medullary cancer may soften and thus lead to a general peritonitis, or dangerous hemorrhage.

In the diffused variety—(infiltrated cancer)—the liver is converted into a white cancerous mass. Atrophy of the liver-cells and obliteration of the vessels and gall-ducts takes place. The liver-cells will be found to have undergone fatty degeneration, and to be colored by bile.

Alveolar or *gelatinous cancer* sometimes extends from other organs to the parenchyma of the liver, and may then transform the whole into a shapeless mass. There may also be cancer of the portal vein.

ETIOLOGY.

Is obscure. Is generally due to cancerous diathesis, occurring principally in males. Is generally preceded by cancer of some other organ.

SYMPTOMS.

Rational.

Obscure at first.
A sense of pressure and fullness in the right hypochondrium.
Symptoms of a local peritonitis (early).
Pain—in the region of the liver (often extending to the right shoulder).
Sensitiveness to pressure.
Tumor—in the right hypochondrium (detected by palpation and percussion).
Ascites—(from compression of the portal vein).
Gastric and intestinal catarrh—(from obstruction to the portal circulation).
Enlarged spleen.
Icterus—(from compression of the large bile-duct and the ductus choledochus, or catarrh of the bile-duct).
Distention of the superficial abdominal veins (an evidence of portal anastomosis, or of severe portal obstruction)
Dyspeptic symptoms.
Fæces—colorless (showing defective secretion or escape of bile).
Urine—peculiarly red or bluish (from abnormal coloring-matter contained).
Cachectic appearance.
Emaciation.

Œdema of the ankles—(from obstructed venous return from the lower extremities).

PHYSICAL.

Inspection.—The ribs are displaced; and the abdomen may be prominent.

Palpation.—The edge of the organ is indurated, with large and small protuberances.

Peritoneal friction may sometimes be felt.

CAUSES OF DEATH.

Dropsy; thrombus of femoral vein; follicular catarrh of the intestines. Thrush in the mouth often precedes death.

TREATMENT.

This being an incurable disease, the treatment at best can only be palliative. *Leeches* may be applied over the region of the liver to alleviate the local peritonitis, and after their removal the surface should be covered with warm poultices to facilitate the flow of blood. The pain should be relieved by *opium*, and the patient supported with good nutritive diet. When ascites occurs, it should be removed by tapping, and this should be done early.

WAXY LIVER.

SYNONYM.

"Lardaceous;" "amyloid."

DEFINITION.

Is due to a deposit of a substance (resembling amylum or cellulose) in the liver-cells, and in the walls of the hepatic vessels.

MORBID ANATOMY.

The liver resembles fatty liver in form; it will appear elongated, flattened, thickened and sharply defined at the edges, hard as a board in consistence, and the peritoneal covering smooth and tense. *On section*, the surface is dry and bloodless, almost homogeneous, and possessing a waxy lustre. If a solution of iodine (Lugol's) be applied to the cut surface, it will assume a peculiar reddish-brown color; after the addition of sulphuric acid, well diluted with

water, there is a violet, and subsequently a blue color produced. There may be frequently fatty degeneration of the organ and also waxy degeneration of the spleen and kidney, occurring at the same time.

On microscopical examination, the liver cells appear round and enlarged and filled with a translucent homogeneous substance. The *middle zone of the lobules* of the liver *are chiefly affected.*

ETIOLOGY.

It occurs in advanced cachexia from scrofulous, cachectic, or syphilitic affections; from mercurialism; caries of bones; tedious suppuration; tubercular phthisis; and it is induced by malaria. There is also a degeneration of the spleen with this disease.

SYMPTOMS.

Enlarged liver without pain (chiefly in scrofulous children).
Ascites—(due to cachexia and the deposit of the amyloid material in the coats of the capillary blood-vessels of the liver, causing portal obstruction).
Spleen—enlarged from waxy degeneration.
Paleness of skin and mucous membranes.
Kidneys affected (producing hydræmia).

PROGNOSIS.

Is unfavorable.

TREATMENT.

The preparations of iodine, especially *syrup. ferri iodidi*, are extensively used; as are also alkaline baths and preparations of *iron.*

GALL-STONES.

SYNONYM.

"Hepatic colic."

MORBID ANATOMY.

The gall-stones may be from the size of a pea to a hen's egg and vary considerably in number. They may be smooth, round, or rough, and some may have convex or concave facets. They are

of low specific gravity, break easily, split when dry, and finally dreak down into dust. They may be white, pale-yellow, dark-brown, green, or black in color. The majority are composed of cholesterin and biliverdin, bile pigment and carbonate or phospate of lime.

There may be slight ulceration of the fundus of the gall-bladder, which may lead to perforation, thus entering the peritoneum and producing peritonitis. The ulceration may possibly communicate with the intestines, or perforate outwardly, after adhesions have formed. The walls of the gall-bladder are often thickened, and undergo cicatrical retraction, and biliary calculi may then be found imbedded in a chalky mass, inclosed in a shrunken and atrophied gall-bladder. Suppurative hepatitis may occasionally be produced.

ETIOLOGY.

Exciting causes.

From foreign bodies in the gall-passages (around which chalk with bile-pigment is deposited); from excess of chalk in the bile (due to drinking lime-water); from lack of, or decomposition of taurocholic acid; from accumulated secretion of bile; from an excess of cholesterin and coloring matter.

Predisposing causes.

Advanced age; female sex; sedentary habits; habitual constipation; over-indulgence in food or drink; cancer of the liver and stomach; catarrh, etc., of the gall-bladder, or ducts, interfering with the escape of bile.

SYMPTOMS.

Begin suddenly and unexpectedly.

Pain—griping, piercing, and intense; extending from the right hypochondrium over the whole of the abdomen to the right side of the thorax and right shoulder. It is often localized at a point on the free border of the ribs when a line drawn from the right nipple to the navel crosses it: and seems to shoot through the abdomen, from that point, to the region of the back.

Patient sighs and moans; is doubled up; rolls about the bed or floor; and presses on the abdomen.

Pulse—small.
Skin—cool.
Face—pale, distorted, and anxious.
Fainting (sometimes) and exhaustion.
Tremblings or chills (spasmodic).
Convulsions of the right half of the body (in rare cases).
Vomiting—obstinate and sympathetic.
Paralysis—has been known to occur.
Permanent closure, inflammation, or ulceration of the excretory bile-ducts may follow and produce symptoms characteristic of these conditions.
Jaundice—slight, and usually precedes the attack.
Constipation—may occur.
Fæces—may be colorless (from want of bile).

CAUSES OF DEATH.

Death may result from biliary obstruction, acholia, marasmus, or peritonitis from perforation of the gall-duct.

TREATMENT.

Karlsbad, Vichy, or any alkaline water should be given which will cause their evacuation, or a preparation of *ether*, ʒ iij.; *oleum terebinthi*, ʒ ij.—dose, ʒ ss. in the morning may be given; the dose should be increased till one pound of the mixture has been taken. *Ether*, ʒ i.; ol. terebinthi, ℈i. may be administered in drop doses; and, in order to relieve the pain, *tincture of opium* (laudanum), gtt. xiij. every hour till sleep is accomplished. *Morph. acet.*, gr. ¼ every hour or two till slight narcotism is produced, has been recommended. *Chloroform* may be inhaled. *Leeches* may be applied in the region of the right hypochondrium, after which warm compresses may be used to facilitate the flow of blood. After the passage of the stone, mild laxatives may prove very beneficial.

HYDATIDS OF THE LIVER.

These usually occur in the right lobe, and may reach the size of a child's head or even larger.

MORBID ANATOMY.

There is a firm, whitish, or yellow fibrous vascular capsule which is adherent to the surrounding tissue. Within this capsule is a delicate cyst, or bladder, consisting of a hyaline concentric layer. The most internal layer of this cyst is studded with minute cells (called the germinal membrane or mother cell). Within this mother cell is a quantity of fluid, consisting of a strong saline solution, and floating in this fluid are the daughter-cells (some thousands).

ETIOLOGY.

From ecchinococci, entering the hepatic lymphatics and developing there (Virchow); from ecchinococci, entering the blood-vessels of the liver and developing there (Leukart); from ecchinococci, entering the bile-ducts and developing there (Friedreich).

SYMPTOMS.

Rational.

Are latent.

A sense of pressure and fullness in the right hypochondrium.
Tumor—in the abdomen yielding an hydatid fremitus.
Jaundice—slight (is usually present as the result of pressure upon the bile-ducts).
Liver—enlarged.
Ascites—(from obstruction to the portal circulation).
Spleen—enlarged.
Gastric and intestinal hemorrhage—(may occur).

Physical.

Inspection—reveals a tumor, which is lobulated and usually of large size if fully developed.

TREATMENT.

Is obscure. They have been opened externally. They may also discharge spontaneously into the intestinal canal, thus assisting in the diagnosis. They may burst into the peritoneal cavity and cause death.

ACUTE YELLOW ATROPHY OF THE LIVER.

SYNONYM.

Black jaundice.

DEFINITION.

Is a combined molecular and fatty degeneration of the organ, and the result of a severe constitutional affection.

MORBID ANATOMY.

The liver will be found sunken against the posterior abdominal wall; it will have a dull yellow appearance; it will be diminished in consistence and also in bulk (occasionally about one-half its original size), and also diminished in thickness and appear flattened. Its serous covering will be loose and often in folds. The gall-ducts and bladder usually contain a scanty mucous secretion; the contents of the intestines are often bloody; the spleen is enlarged; and eccyhmoses are frequent, especially in the peritoneum and gastric and intestinal mucous membrane.

ETIOLOGY.

From continued biliary obstruction producing distension of the bile-ducts from an excessive formation of bile causing continued pressure on the liver-cells and consequent atrophy; from intemperance; from venereal excesses; from constant exposure to malarial poisoning, etc. Is a very rare affection, never occurs in childhood, more frequently in women and especially during pregnancy.

SYMPTOMS.

In the first stage.

Anorexia.

Pressure and *fullness* in the epigastrium.

Jaundice—(hepatogenous, but not in all cases).

In the second stage.

Jaundice—which increases (hematogenous)

Liver—very *sensitive to pressure.*

Headache—severe.

Restlessness and *delirium* (sometimes).

Muscular twitchings—may occur (from extention of excitement to the motor nerves).

Depression and lassitude—excessive.

Temperature—very high.

Pulse—frequent and subsequently small.

Tongue and gums—dry, and covered with sordes.

Involuntary evacuations.

Hæmatamesis—may occur from destruction of the capillaries.

Hemorrhage from the bowels—may occur.

Deep sleep which gradually passes into a *profound coma.*

Profuse perspiration.

Death—usually from the second to the fifth day after the coma commences.

Physical examination shows very rapid *decrease of liver dullness,* the sole pathognomic symptom of the disease.

Percussion sound—is at first very full in the epigastrium due to the diminution in the size of the liver commencing in the left lobe. but frequently no trace of liver dullness can be found after a few days. There is an increase in splenic dullness which is important but does not always occur.

DIFFERENTIAL DIAGNOSIS.

From acute hepatitis—from catarrhal jaundice.

PROGNOSIS.

The disease is generally a fatal affection.

TREATMENT.

Medication is hopeless unless the disease be detected in the first stage when saline cathartics may be administered, leeches applied at the anus, and cold compresses over the right hypochondrium.

DISEASES OF THE BRAIN AND NERVOUS SYSTEM.

CEREBRAL HYPERÆMIA.

ETIOLOGY.

Active or Fluxionary Hyperæmia.

Increased Heart's Action.

From augmented energy, as in fevers, or bodily or mental excitement.—From cardiac hypertrophy, (as an independent disease).—From cardiac hypertrophy accompanying an obstructed circulation (compensatory). From too slight resistant power of afferent blood-vessels whether congenital or acquired ("Rush of blood to the head").

Collateral Fluxion or increase of Lateral Pressure in the carotids.

From obstructed escape of blood from the aorta into other branches.—From conatrctions or closure of aorta (due to compression of adominal aorta, and its branches, from distended intestines and exudations).—From obstruction of cutaneous circulation, as in cold stage of intermittent fever.—From severe cold.—From severe muscular exercise, by pressure of muscles on the capillaries.

Paralysis of Vaso-motor nerves (of cerebral vessels).

From section of cervical portion of the sympathetic.—From use of spirituous liquors.—From poisons.—From excessive mental emotions and activity (especially).—Atrophy of the brain following apoplexy.

Passive or Congestive Hyperæmia.

Compression of the Jugulars or Superior Vena Cava.

From strangulation.—From enlarged thyroid glands.—From enlarged lymphatic glands, affecting the jugulars.—From aneurism of the aorta.—From enlarged glands, affecting the superior vena cava.

Energetic Expiratory Movements with Glottis contracted.

From coughing, straining, playing wind instruments, etc.

Non-compensated Valvular Lesions of Heart.
From capillary hyperæmia (since over-filling of the veins obstructs the flow from the capillaries).—From impeded return of venous blood.—From valvular disease of right heart, preventing escape of venous blood, and increasing amount of blood in skull.—From valvular disease of left heart (causing delayed circulation in the lungs).

Lung Affections (interfering with circulation).
Emphysema (especially last stage), producing general cyanosis of brain.—Pleuritic effusion.—Cirrhosis of lung.

Plethora (by increasing the amount of blood).
From a too free supply of food and drink (having a tendency to apoplexy).

SYMPTOMS.

Headache—more or less severe (due to irritation of filaments of the fifth nerve going to the dura mater).
Feeling of constriction in the head.
Great sensitiveness to bright lights and loud noises.
Flashes before the eyes.
Tinnitus aurium.
Dizziness.
Nervous irritability or depression—patient is annoyed by the slightest irritation.
Face and conjunctiva—may be reddened.
Temperature—may be elevated.
Pulse—full and rapid.
Hallucinations and illusions.
Respiration—retarded.
Sense of formication in the lower extremities.
Slowness and sluggishness in the movements of the patient.
Delirium—(in the asthenic type).
Fullness of the meningeal veins.
Insomnia or disturbed dreams.
Restlessness.
Vomiting.
Convulsions—may occur, with loss of consciousness.
Paralysis.

PROGNOSIS.

Depends upon the cause.

TREATMENT.

As this is simply a symptom which accompanies so many diseases, the cause must therefore be treated. General and local blood-letting, cold to the head and derivation to the skin and bowels have enjoyed a good reputation, yet, neither the one nor the other must be used indifferently. Should there be collateral fluxion to the brain, the obstructions to the circulation, by which the pressure of blood in the carotids is increased, must be removed. In adults, suffering from constipation, headache, tinnitus aurium, etc., and especially in children, where constipation is accompanied by convulsions, evacuation of the intestines by laxatives or enemata of vinegar and water frequently has a wonderful effect. Should these means prove insufficient, depletion in the adult, and *leeches* applied to the head in children, should be resorted to if symptoms of depression occur.

In congestive hyperæmia, resulting from compression of the jugular veins or vena cava, *venesection* or *leeches* behind the ears may be resorted to if the obstruction to the current of blood cannot be removed.

ANÆMIA OF THE BRAIN.

MORBID ANATOMY.

If dependent upon embolism, emboli are almost always found in the left fissure of Sylvius (producing hemiplegia during life). The brain is discolored, and the gray substance is paler than normal, and softened in the region of the embolus.

ETIOLOGY.

It may occur from spasmodic contraction of the arteries, or be due to obstruction to the carotid and vertebral arteries (by an embolus or as the result of thrombosis): to obstruction to the arteria fossæ Sylvii (by an embolus or thrombus), causing obstruction in the circle of Willis; to obstruction from pressure, as in

the case of a tumor, or the presence of œdema around an apoplectic clot, or a point of inflammation, or softening; from compression of the capillaries by extravasation of blood, tumors, etc.; and from diseases contracting the space within the skull, as exostosis, cancer, etc.

SYMPTOMS.

Premonitory symptoms.

Disturbance of the circulation (probably due to the contraction of cerebral vessels, or to senile cerebral atrophy).
Pain—in the head.
Dizziness.
Tinnitus aurium.
Flashes appear before the eyes.
Loss of memory and power of thought.
Patient is apathetic and indifferent.
Inclination to sleep (which is disturbed and uneasy).
Aphasia—which is usually of the amnesic variety (in case of an embolus of the left middle cerebral), and hemiplegia.
A sense of formication, or of certain limbs going to sleep.
Contraction or paralysis of the limbs.

Of disease of the posterior cranial fossa.

Pains—in the back of the head (from filaments of the fifth nerve to the tentorium cerebelli).
Sympathetic vomiting.
Peculiar dizziness (when moving about).
Diminution of sensibility and motor power (but not complete paralysis).
Impaired articulation and deglutition.

TREATMENT.

Promises very little, if any benefit, should thrombosis and embolism occur, as therapeutic remedies cannot remove the obstruction, and absorption is all that can be hoped for. Should there be no symptoms of irritation, a strengthening and stimulating treatment is indicated; but, if there be symptoms, such as severe headache, contractions, etc., *cold* has been suggested, and also the application of *leeches* behind the ears.

APOPLEXY.

DEFINITION.

A condition characterized by complete functional inactivity of the brain; produced either by swelling of the vessels (miliary aneurism) or extravasation of blood into the brain-substance. Symptoms of a similar character may be produced by serous effusions, or by organic diseases of the brain.

VARIETIES.

Sanguineosa; serosa; and nervosa; the first being the most common.

MORBID ANATOMY.

There may be small, numerous, closely packed effusions, which denote capillary rupture, or a larger quantity of blood, denoting rupture of the larger vessels. If it should result from capillary rupture, the brain-substance is dotted over with dark-red punctate extravasations; it may cause the brain-substance to appear of a yellow or reddish color, and to become moist, relaxed, or broken down (causing red softening). Small clots press on the brain filaments, but large clots break up and mix with the brain-substance. The blood may escape into the ventricle, or even in the sub-arachnoidean space. The most frequent seats in which the clots are found are in the corpus striatum, the optic thalamus, the peduncle of the brain, and the large medullary masses of the hemispheres. The clot, which varies from the size of a hemp-seed to that of a man's fist, consists of blood and broken-down brain-substance, which may be either fluid or partly coagulated. The clot is often inclosed by a new formation of connective-tissue, starting from the neuroglia, there is also a delicate connective-tissue which surrounds the walls and traverses the clot. After a time a cyst forms, inclosing the clot, and the serum may ultimately be absorbed. The injuries to the brain-substance often produce a gradual diminution of mental power, said to be due to atrophy

of the brain-substance, associated with degeneration, extending into the spinal medulla.

ETIOLOGY.

It occurs principally from rupture of the smaller arteries or capillaries from structural disease (endarteritis); from increased pressure (due to a fatty degeneration of the walls of the arteries), in cachectic, badly nourished, chlorotic persons (producing fragility of the vessels); from small dissecting aneurisms; from weakness of the cerebral vessels (as in typhus fever and scorbutus); from gradual atrophy of the brain-substance; from plethora (where there is a morbid fragility of the vessels); and from increased pressure and existing atheromatous changes.

It may occur at any age and time of the year, and without any appreciable existing cause.

SYMPTOMS.

Premonitory symptoms.

Headache—or fullness of the head.
Tinnitus aurium.
Flashes before the eyes.
Dizziness.
Insomnia.
Formication
Irritability.
} due to general or partial hyperæmia of the brain.

Symptoms of attack (modified by seat and extent of hemorrhage).

Fit—(which is due to anæmia of the brain-substance from sudden compression of the capillaries), comes on at the onset, or may not come on till after paralysis has occurred, or it may come on suddenly with coma, or rapidly developing coma.

Breathing—loud and stertorous.
Respirations—diminished.
Vomiting—at the commencement of the attack.
Pulse—very slow.
Pupils—irregular.
} symptoms of cerebral compression.

Pulsation of the carotids—more marked than in health.

Aphasia—(of the amnesic type) may occur, if Broca's centre be involved (3d frontal convolution).

Temporary paralysis of muscles, or any group of muscles—may exist, (the various types of monoplegia).

Paralysis of the arm and leg of one side (hemiplegia)—from destruction of the corpus striatum, optic thalami, or peduncles of the brain on the opposite side, by a clot.

Loss of will and previous capabilities of thought.

Facial paralysis of Bell—where the corner of the mouth hangs down on the affected side, and is raised on the sound side—Tongue, if protruded, goes toward the paralyzed side.—Eye of the affected side cannot be closed.

Anæsthesia—from compression of the sensory nerve fibres of the brain (posterior third of internal capsule).

General convulsions—(may occur) if the clot acts as an irritative lesion to the motor area of the cortex, and does not affect the deeper parts.

Death—may result from hemorrhage into the pons, if the hemorrhage be of considerable amount; from hemorrhage into the medulla, if the hemorrhage be slight.

Instead of improvement in twenty-four hours, the symptoms usually increase, if the clot be large or the damage to the brain be extensive.

DIFFERENTIAL DIAGNOSIS.

From embolism; cerebral softening; and all causes of coma.

PROGNOSIS.

This depends on the extent of the extravasation and its seat. One attack is, usually, soon followed by another, since the vessels of the brain are diseased.

TREATMENT.

During the attack—venesection may be performed, but only if the heart-impulse be strong and the sounds loud, with a regular pulse and no œdema. *Stimulants* should only be administered if weak heart and irregular pulse occur. *Mustard plasters* may be applied to the chest and calves of the legs. The skin should be rubbed vigorously. Dash cold water on the breast, or drop melted sealing wax on the chest (Niemeyer).

After the fit.—The diet should be mild. The bowels should be

kept open. Cold should be applied to the head, *leeches* behind the ear, and the *induced current* to the paralyzed muscles.

MENINGITIS.

VARIETIES.

See page 9.

ACUTE MENINGITIS.

SYNONYM.

"Simple meningitis of the convexity."

DEFINITION.

Is an acute form of inflammation of the coverings of the brain.

MORBID ANATOMY.

On removing the calvaria, it will be found to be adherent to the dura mater. The brain-substance will be of a greenish color, due to the infiltration of serum, fibrin, and pus into the meshes of the pia mater. It is most marked at the convexity and along the course of the vessels. The dura mater will be found thickened and slightly adherent to the calvaria. The arachnoid is opaque, its surface roughened, slightly adherent to the dura mater, and covered with punctate spots. The cerebral vessels and their branches are injected and prominent. The ventricles will be found empty. The brain-substance will be softened, in various parts, from the pressure of the exudation. The sulci will be deepened and the convolutions sharpened.

ETIOLOGY.

It usually occurs between the ages of 18 and 20, and 40 and 50, and is more frequent in men than in women. It is rarely an independent disease; but is usually secondary to some injury, diseases of the skull, and dura mater, or other diseases. It occurs in cachectic persons from long exhaustive illness. It may occur in pneumonia, pleurisy, acute exanthematous and infectious diseases

(typhus especially, from the tendency toward cerebral engorgement), and Bright's disease; from exposure of the head to the rays of the sun, or very low temperature; from the misuse of liquors; from caries of the petrous portion of the temporal bone, or from over-taxation of the mental faculties.

STAGES.

1. *Headache.* 2. *Delirium.* 3. *Coma.*

SYMPTOMS.

Stage of headache (lasts from 24 to 72 hours).

Rarely ushered in with a chill.
Headache—is severe and increases.
Photophobia.
Pupils—contracted (due to congestion of the brain, producing reflex manifestations).
Pulse—elevated (120 to 140), small, hard, and regular.
Abnormally acute sense of hearing.
Intolerance to noises.
Nausea and vomiting (possible).
Constipation or diarrhœa (may occur).
Tongue—moist and coated.
Gait—unsteady.
Face—pale at first, but afterward flushed.

Stage of delirium (lasts from a few hours to three days).

The delirium may be muttering or active.
Patient is constantly trying to get out of bed.
Talks to imaginary objects or persons.
Pupils—unequally contracted.
Pulse—frequent, intermittent, or irregular.
Constipation.
Insomnia.
Alternating flushes and pallor of the face.
Convulsions (may occur).
Patient becomes gradually stupid, and after three days coma usually takes place.

Stage of coma.

Pulse — slow 40-60 (in the first stage, afterward becoming very rapid).
Temperature—may fall below normal, but rapidly rises.
Loss of sensation and voluntary motion.
Delirium—may be slight and muttering.
Pupils--dilated (from inter-cerebral pressure) in this stage.
Flapping of the cheeks (in the first part of the coma).
Face—turgid.
Constipation.
Eyes—wild and brilliant.
Sphincters—may be relaxed.
Blueness of the fingers and lips.
Profuse perspiration.
Retention of urine.
Capillary circulation—poor.
Respiration—sighing and marked.
Heart's sounds—indistinct.
Tongue--dry and brown (from not breathing through the nostrils).
Patient passes into collapse, finally resulting in death.

DIFFERENTIAL DIAGNOSIS.

This disease may be confounded with typhus fever; acute uræmia; small-pox (for the first few days); or delirium tremens.

In typhus fever--the delirium comes on late in the disease: the pulse will be frequent and small; and the countenance dull and heavy.

In acute uræmia--there will be the small amount of urine, containing albumen and casts; and the pulse will be soft and slow.

In small-pox—you will have pain in the head, back and loins, the eruption occurring along the roots of the hair, and at the alæ of the nose after thirty-six hours.

PROGNOSIS.

Is unfavorable, especially when pus, serum, and lymph are deposited in the arachnoid.

TREATMENT.

Various methods have been suggested, but it has been found of late years that the best treatment is to keep the patient quiet in bed, and relieve the pain by *hypodermics of morphine.*

SUB-ACUTE MENINGITIS.

MORBID ANATOMY.

In this form of meningitis no pus is exuded, and the cell exudation is slight, but there is a sero-fibrinous exudation under the arachnoid into the meshes of the pia mater. Floccules of lymph will also be found along the line of the vessels. The arachnoid will have lost its brilliancy and become opaque and thickened, and when held up to the light, white spots may be seen, chiefly in the lines of the vessels, together with spots of ecchymosis. The vessels may be engorged. Congestion may occur, but this will depend upon the amount of serous effusion present. Effusion into the ventricles may occur. *It should be borne in mind that no effusion is inflammatory unless it be found to contain fibrin, and the arachnoid be thickened and opaque, with little spots of ecchymosis and enlargement of the vessels.*

ETIOLOGY.

It may be secondary to Bright's disease; typhus fever; malaria; or any exanthematous disease. When it occurs in Bright's, the slow pulse usually precedes the coma.

STAGES.

Same as those of the acute form.

SYMPTOMS.

Stage of Headache.

Is not present, as a rule; or if it should be, is not prominent.

Stage of Delirium—(lasts from a few hours to one or two days).

Delirium—is not active, but stupid and senseless. Patient gets out of bed and is easily put back again, and then gets out again after a short while.

Stage of Coma (comes on rapidly).

Pulse—slow, at first, afterward becoming very frequent (due to a failure of heart's power).

Respiration—sighing.

Tongue—dry, dark-brown.

Pupils—dilated.

Collapse and death.

PROGNOSIS.

About one-third of the cases recover. Should it be due to Bright's disease, it will be progressive, and terminate in death. If to pyæmia, it will be slowly fatal.

TREATMENT.

Should it occur in connection with any of the fevers, the patient should be kept in bed, with the head slightly elevated. Only one person should be in attendance, and the room kept perfectly dark and quiet. *Counter-irritation* should be applied to the feet in the form of a mustard foot-bath; the feet should be kept in it for about one hour or until redness of the surface is produced. Should there be constipation, *cathartics* are indicated. Blisters and counter-irritants may be applied to the back of the neck. *Stimulants* may be administered freely if it occur from typhoid or typhus fever. *Iodide of potassium* (in gr. xx. to xxx. doses) every three hours are especially indicated should syphilis be suspected.

CHRONIC MENINGITIS.

SYNONYM.

"Pachy-meningitis." Is termed *externa* and *interna* (or hæmatoma of the dura mater) according as it may effect the external or internal surface of the dura mater.

MORBID ANATOMY.

In the externa—the dura mater is thickened and opaque (from an increase of connective-tissue development) and adherent to the cranium. It is red and vascular. Pus will be found between the dura mater and the skull. Pia mater may be greatly inflamed.

In the interna—there may be hæmatoma (resulting from inflammation) between the dura mater and the cranial bones, having its origin in the dura mater or underneath the arachnoid. The hæmatoma is produced by rupture of the small vessels in the new connective-tissue. There may be a destruction of the arachnoid and the formation of abscesses containing three or four ounces of pus, resulting in pyæmia.

ETIOLOGY.

Advanced stage of syphilis; caries or fissure of the petrous portion of the temporal bone; suppuration of the orbit; extension of inflammation from the internal ear; chronic alcoholism; chronic Bright's disease.

SYMPTOMS.

Present in all varieties.

Are often vague.

Pain in the head—which is localized, and increased by exercise, mental labor. excitement, etc., may usually be detected, at the vertex and forehead.

If due to alcoholismus.

The vision and special senses are disturbed.

Tinnitus aurium.

Gait—unsteady.

Hyperæsthesia or anæsthesia.

Delirium - slight.

Talks—incoherently.

Sleep is disturbed, at first; then the patient becomes disposed to sleep and gets stupid.

Inequality of the pupils—(or may be contracted).

Slight facial paralysis.

If due to otitis media or acute otorrhœa.

Perforation of the tympanum and discharge of pus from the ear.

Slight stiffness of the muscles at the back of the neck.

Pupils—slightly irregular.

Vomiting.

Temperature—slightly below normal.

Pulse - about 80 or a little below normal, and may be intermittent or irregular on movement.

Peculiar expression of countenance—(pyæmic in character).

Skin—pale, dingy yellow.

Face—puffed.

Delirium of an hysterical type—may occur.

If from syphilis.

Patient wanders, gets out of bed, and there may be facial paralysis (from inter-cerebral pressure).

In all cases having cerebral symptoms assuming an hysterical type, with an intermittent pulse, and discharge from the ear, chronic or pachy-meningitis may always be anticipated.

PROGNOSIS.

Is usually unfavorable.

TREATMENT.

Is usually hopeless, if the disease be extensively developed.

ACUTE HYDROCEPHALUS.

SYNONYMS.

Tubercular or basilar meningitis, or "dropsy of the brain."

MORBID ANATOMY.

There is congestion on the surface of the brain, with a serous effusion extending into the ventricles. At the base of the brain, there is a sero-fibrinous exudation (sometimes sero-purulent) which is most marked at the optic chiasm and fissure of Sylvius. The arachnoid is thickened and opaque, and the dura mater is also opaque. The convolutions are flattened, and the sulci deepened. Nodules will be found in the meshes of the pia mater (tubercle), both of the brain and spinal cord which vary in size from that of a pin's head to a pea, and which later on, may undergo degeneration.

The *characteristic lesion* is an effusion of serum and lymph, and a tubercular deposit at the attached surface of the arachnoid

(most marked at the optic commissures and island of Reil) at the base of the brain along the fissures, causing thickening and extravasation into the pia mater.

ETIOLOGY.

It occurs in children of a scrofulous diathesis between two and five years of age; in old age; in alcoholismus; and from bad food and air; and want of cleanliness. This disease usually lasts from ten to fourteen days or even three weeks.

STAGES.

This disease has three stages:—1st. *Delirium.* 2d. *Coma.* 3d. *Collapse.*

SYMPTOMS.

Premonitory symptoms—(of great diagnostic value).

Change in the disposition of the child, stops in his play, and wants to be held.
Rests his head on the mother's knee or on his own hand.
Gnashes his teeth in his sleep.
Thumbs—flexed on palms of the hand during sleep.
Ptosis (the upper lid falling, giving the eye the appearance of being half closed).
Head thrown back and bored into the pillow.
Moans during sleep and rolls the head from side to side.

In the stage of delirium.

Patient is fretful and uneasy.
Pains in the head—patient puts the hand to the head.
Pupils—contracted.
Photophobia.
Patient answers slowly but rationally.
Vomiting—is projectile.
Jactitations.
Pulse—firm, hard, and regular (110 to 140); can be accelerated by very slight excitement.
Abdomen—retracted.
Temperature—may be remittent or normal.
Respiration—sighing.

In the stage of coma.

Disposition to sleep—(after two or three days).
Involuntary evacuations.
Pupils—do not respond to light (as at first).
Pulse—becomes slow (60 to 70).
Hydrocephalic cry.
Convulsions—probable.
Eyebrows—contracted.
Slight paralysis—(may occur).
Picking at the bed-clothes.
Head rolls from side to side.
Lethargy - increases gradually.

A strange remission may occur before the third stage, wherein the child may appear to be recovering; it is only temporary, however, as the patient passes into a deeper coma.

The stage of collapse.

Pulse—increases (160 to 200), small, frequent, intermitting, and not easily compressed.
Paralysis—may occur of one arm or leg, or even hemiplegia may be developed.
Strabismus.
Sphincters—relaxed.
Eyes—rolled up.
Head—rolled to one side.
One-half of the body may be hot, and the other half cold.
Pupils—irregularly dilated.
Inspiration—rapid.
Expiration—imperfect.
Imperfect capillary circulation.
Death—frequently occurs with convulsions.

PROGNOSIS.

About seven-eighths of the cases die, and those who recover are usually crippled.

DIFFERENTIAL DIAGNOSIS.

It may be confounded with infantile remittent fever; Bright's disease (in children); spurious hydrocephalus.

TREATMENT.

The patient should be kept in a dark, quiet, well-ventilated room. The diet must consist of milk, or milk and lime-water. A piece of ice should be put in a bladder and laid on the pillow a short distance from the head. The feet should be placed in a mustard foot-bath. *Blisters* may be applied early behind the ears. *Iodide of potassium* may be given in full doses, but it has no effect. Small doses of *morphine* (gr. $\frac{1}{24}$) have been recommended by Hasse. In order to prevent this disease if possible, especially if the parents are of a tubercular diathesis, the child should be sent at once into the country and kept there for at least *two* years. A good strong wet-nurse should be obtained, or else the child should be fed on milk from the bottle for the first eighteen months; no mixed diet should be allowed. *Cod-liver oil* should be given continually especially during the cold months. Care should be taken to see that the child is properly clad. If at the end of two years the child begin to be fretful, the whole habit of life should be immediately changed.

CHRONIC HYDROCEPHALUS.

VARIETIES.

1st. Intra-uterine. 2d. When it occurs after birth, before closure of the sutures. 3d. When it occurs some time after birth, when the sutures have closed.

MORBID ANATOMY.

There is failure of the fontanelles to close; enormous growth of the cranium quite out of proportion to the face; separation of the sutures; and fluctuation between the fontanelles. There is an accumulation of fluid in the ventricles and in the meshes of the pia mater. It is said to be external when the fluid is in the meshes of the pia mater, and internal, when the fluid is in the ventricles.

SYMPTOMS.

First Variety.

The head is not much increased in size, but is of a peculiar shape.

The child is often weak-minded—as it has a very small amount of brain.

Second Variety.

The head increases in size.

Eyes—sunken.

Fontanelles—protruding; fluctuation may occur between the anterior and posterior fontanelles.

The child is precocious for the first year or two, when nervous symptoms appear, and death usually occurs about the age of four or five years.

Third Variety.

Gradual growth of the cranium out of proportion to the face.

Gait—unsteady.

Paralysis—in some cases (from inter-cerebral pressure).

Involuntary evacuations.

Child may reach adult life, but it is always a misery and trouble to every one.

TREATMENT.

Tapping, bandaging, counter-irritation, and various other methods have been tried, but all have amounted to nothing.

CEREBRO-SPINAL MENINGITIS.

SYNONYM.

"Spotted fever."

DEFINITION.

Is an infectious disease, characterized by an inflammation of the meninges of the brain and spinal cord. It may be of an epidemic or sporadic type.

MORBID ANATOMY.

On opening the skull, there will be seen a green exudation on

the surface of the brain, which is most marked at the base, and which extends down the cord. The sulci will be found to be deficient and the convolutions sharpened. The dura mater will be tense and covered with hemorrhagic deposits. There is an exudation extending into the meshes of the pia mater of the brain and spinal cord, which may be fibrinous, sero-fibrinous, or sero-purulent in character, and is very plentiful in the region of the optic chiasm, fissure of Sylvius, base of the cerebellum, and the fissures of the cerebrum embedding the nerves.

ETIOLOGY.

The attacks are more frequent in the winter than in summer, as the disease disappears as the summer comes on. It occurs most frequently in children (from 12 to 20) and middle life, and is extremely rare in the aged. Bad hygienic surroundings are great predisposing and exciting causes to this disease, especially of the epidemic form.

SYMPTOMS.

Chill or rigors.
Pain—in all the bones.
Headache—which is severe and increases.
Photophobia.
Pupils—contracted.
Jactitations.
Nausea—and perhaps vomiting.
Pulse—80 to 100, hard, wiry, and increasing—later on may be slower than normal.
Temperature—moderate.
Respirations—30 to 40 a minute and increasing.
Countenance—pale and anxious (associated with knitting of the brows).
Eruption—appears early on the mouth, cheeks, eyelids, ears, and occasionally on the extremities (but that depends upon the severity of the case). Is usually herpetic in character.
Delirium—slight.
Pain—in the back of the head and along the spine.
Muscles of the neck—are rigid.

Opisthotonos—may occur.
Thighs may be flexed on the trunk and the leg on the thigh.
Arm is drawn across the chest, fore-arm is flexed on the arm, and the fingers are clinched.
Abdomen—is first swollen and then shrunken.
Great sensitiveness of the skin.
Deafness—(may occur)—from inflammation of auditory nerve.
Blindness—may occur from keratitis.
Coma—may occur (when the following symptoms will be noticed):
Pupils—unequally dilated.
Respiration—sighing.
Urine—is passed involuntarily, or may have to be drawn off.
Constipation or involuntary evacuations—may occur.
Capillary circulation is imperfect.
Patient is aroused with difficulty, and may possibly take food.
Eruption—may appear only in this stage.
Emaciation—is rapid.

CAUSES OF DEATH.

Death may occur from acute œdema of the lungs, bronchitis, or pneumonia. Any of these may occur on the first or second day, but usually about the fourth or fifth day. In some cases, death appears in a few hours after the symptoms have set in, from paralysis or marasmus.

COMPLICATIONS.

1. Croupous pneumonia—with no rational complications.
2. Croupous nephritis—from the cerebro-spinal axis affecting the parenchyma of the kidney.
3. Œdema and congestion of the lungs—due to paralysis of the muscles of the chest.

SEQUELÆ.

1. Impairment or loss of hearing and speech.
2. Impairment or loss of sight—due to inflammation of the retina, or from corneal ulcer.
3. Chronic hydrocephalus (especially in children).
4. Paralysis and great emaciation.

5. Neuralgic pains—about the head in the region of the cranial nerves.

DIFFERENTIAL DIAGNOSIS.

It may be confounded with other forms of meningitis; with pneumonia in children. Sporadic cerebro-spinal meningitis may be mistaken for tubercular meningitis.

The sporadic form is ushered in by a chill and rapid pulse, whereas, in tubercular meningitis, there is no chill, and the pulse is slow at the beginning.

PROGNOSIS.

Is uncertain. The character of the epidemic must be taken into account.

In severe cases, the prognosis is bad. In mild cases, recovery is possible.

TREATMENT.

It is the custom among the Germans to apply ice-bags to the head and spine, while others prefer hot applications. *Quinine* is of little or no use; it will not even lower the temperature. The patient should be kept in a large, quiet room. The best and largest quantity of the most nutritious concentrated diet must be given. The temperature should be reduced by sponging the surface; and the only remedy that can be employed is *opium* (atropine, gr. $\frac{1}{60}$, and morphine, gr. $\frac{1}{3}$) hypodermically; the patient must be kept thoroughly under its influence until death or recovery ensue.

EPILEPSY.

SYNONYMS.

Falling sickness; morbus sacer; *haut* or *grand mal;* and *petit mal*.

DEFINITION.

Is a chronic disease, characterized by a complete loss of consciousness and sensibility, accompanied, in the severe type, by a sudden falling, and distortion of the countenance, and convul-

sions. *Petit mal* is characterized by incompleteness of the above symptoms, viz.: no convulsions, only twitchings; it is also termed epileptic vertigo.

MORBID ANATOMY.

Often shows asymmetry, such as imperfect development of the skull; diffuse thickening or exostoses; the dura mater may be thickened, adherent, and calcified; tumors, or deposits in the brain; chronic hydrocephalus; cerebral hypertrophy; changes in structure of the spinal cord and medulla; sometimes alterations in the substance of the brain; scars, etc.

ETIOLOGY.

Excitement of the motor nerves, proceeding from the medulla oblongata and the portion of the brain lying upon the base of the skull; from dilatation of the arterioles and capillaries of the medulla, with thickenings of their walls, producing irritation; from cutting off the blood supply (anæmia), or causing afflux of arterial blood to the medulla; tumors pressing on the peripheral nerves; cerebral tumors and other cerebral diseases; a morbid state of the medulla; venous engorgement and over-charging of carbon dioxide, from spasmodic closure of the glottis; from insanity, intemperance, cachexia, onanism, mental emotion, violent fright, wounds, syphilis, worms in the intestines, etc.; or, it may be hereditary, often affecting several generations.

It occurs particularly in females, from infancy up to thirty years of age.

SYMPTOMS.

Are usually ushered in by

Aura epileptica—may appear as a sense of vapor arising from the extremities toward the head; or like a creeping, warm, or numb sensation; or like a darting pain in the brain; or like peculiar odors.

Aura motor—resembles twitchings or palsy.

Aura mental—like hallucinations; sparks or colors appearing before the eyes; or tinnitus aurium; or dizziness.

Patient utters a shrill cry.

Loss of consciousness and sensation.

Convulsions.
Tonic contractions—the patient may fall backward, sideways, or forward.
Head—is drawn to one side.
Mouth—is firmly closed.
Face—cyanosed.
Eyes—wide open, rolled upward or inward.
Pupils—dilated.
Thorax—fixed.
Respiratory movements—arrested.
Jugulars—distended.
Clonic spasms and convulsions.
Grating of the teeth.
Tongue—is bitten.
Foaming at the mouth.
Blood mixed with the foam (from biting of the tongue) appears at the mouth.
Thumbs—turned in on the palms of the hands.
Head—jerked from side to side.
Heart's action—accelerated.
Pulse—small and irregular.
Skin—bathed in perspiration.
Involuntary evacuation of the bladder and rectum.
Involuntary erections and seminal emissions.

PROGNOSIS.

Depends upon the cause. Some cases recover perfectly, while others withstand all remedial measures. The epilepsy of infants and young children gives a better prospect of recovery than when developed late in life.

TREATMENT.

No epileptic mother should ever be permitted to nurse her child; a vigorous wet-nurse should be obtained, and their home should be in the country. Regulate the system and surroundings. Forbid mental occupation of any kind. The only condition of success is patience, both on the part of the physician and on that of the patient. It is necessary to be very stern as regards excessive

sexual intercourse, onanism, drink, etc. If anæmia occur, nourishing diet, wine, iron, and moderate exercise in the open air, are indicated. If it occur from scars, foreign bodies, etc., etc., they must be removed. The following methods of treatment have been suggested, with more or less beneficial results, viz.: setons, moxa, pustulating ointments, cupping, blisters, etc.; four leeches applied to the back of the neck at a time, every fortnight or three weeks; or ℞ atropine, gr. ij.; sp. vini rect., ʒ iiss ; commence with gtt. i. every two or three hours, and increase till gtt. xx. are given at a dose; or, gtt. v. nitrite of amyl pearls; these are crushed on the handkerchief just as an attack is coming on, and the patient made to inhale it.

Bromide of potash, in large doses, or *bromide of sodium* (this latter is three times as strong as br. potash).

Preparations of *oxide of zinc* or *arsenic*.

When it is complicated with syphilis, *iodide of potash* or *iodide of lime* will be found serviceable. *Iodide of lime* comes in whitish-yellow cakes, and must be kept from the light, because it deliquesces and becomes brown, on account of the iodine.

CEREBRAL SOFTENING.

VARIETIES.

(1) White or non-inflammatory. (2) Red or inflammatory. (3) Yellow.

DEFINITION.

Is a disease characterized, during life, by impairment of the mind, sensibility or mobility, and after death by softening and degeneration of the cerebral substance.

MORBID ANATOMY.

The cerebral substance is a moist, gelatinous, trembling pulp, which may be of a white or grayish-white color, or, possibly, red or yellow. The yellowish color is said to depend on capillary hemorrhage or fatty degeneration (is due to infiltration of the

disintegrated brain-substance with escaped and altered coloring-matter of the blood, or to a close aggregation of fatty matter).

ETIOLOGY.

Is due to necrosis (resulting from obstruction of the vessels and insufficient development of the collateral circulation, or to local inflammatory changes); occurs generally in the greater hemispheres, chiefly in the medullary substance. It may follow a rupture of the cerebral arteries; this latter occurs chiefly in the aged and decrepit, from disease of the vascular walls, which generally induces thrombosis.

SYMPTOMS.

Peripheral arteries—rigid, turgid, and contracted (in the old).
Large arteries—dilated (if atheroma exist in the vessels).
Impairment of mind, sensibility, and mobility.
Paralysis—which may precede or follow the mental impairment).

PROGNOSIS.

Is unfavorable, unless dependent upon some local cause which can be overcome.

TREATMENT.

Same as in anæmia of the brain, (page 228).

MYELITIS.

DEFINITION.

It is a red softening of the spinal cord, in circumcribed spots; which becomes yellow, after a lapse of time, from fatty degeneration of the broken-down nerve-elements; it may produce abscess of the spinal cord. It may be either acute or chronic.

MORBID ANATOMY.

A cavity may be formed in the spinal medulla filled with serum, and traversed by connective-tissue; or there may be an induration from connective-tissue proliferation. *On section*—the gray sub-

stance of the cord is darker, redder, and less consistent than normal. The medulla is swollen, and in its centre is a reddish, rusty, or yellow pulp. The tissues are broken down and often absorbed, so that there may be a cavity filled with serum.

ETIOLOGY.

Inflammation extending from the vertebra to the membranes and thence to the medulla; curvature of the spine; wounds; contusions; syphilitic exostoses; cold; sexual excesses; excessive straining; dissolute habits; suppression of perspiration of the feet; masturbation.

SYMPTOMS.

Severe fever—at the onset.
Extensive pain in the back and extremities.
Tetanic spasms of the neck.
Clonic spasms of the extremities.
Dyspnœa—(if the disease be situated very high up).
Paraplegia and anæsthesia (partial).
Numbness and irritation.
Vague pains.
Sensation of pricking.
Formication.
Painful contraction of the extremities.
Spasmodic twitchings—due to irritation from inflammation of the motor nerves.
Pain—*like a cord tied round the waist*—dull at the point of inflammation. This pain *is increased by pressure on the spinous processes and not by the movement of the spinal cord.*
Paralysis—becomes complete; the bladder and rectum are paralyzed, and atrophy of the paralyzed muscles takes place—heaviness and helplessness of the lower extremities.
Urine—will be alkaline. Digestive powers—are good, unless the disease is high up in the spinal cord.
If *cervical region be affected* — then the upper extremities and and respiratory muscles are involved.
If *the dorsal region be affected*—then the sphincters may be involved.

If *the lumbar region be affected*—then the lower extremities are involved.

SYNOPSIS OF SYMPTOMS.

Gradual paralysis—extending upward.
Pain—in the back on pressure, (not increased on motion of spine).
Sense of heat on the affected portion, whether heat or cold be applied.
Sensation as if a tight cord were tied round the body, corresponding to the seat of disease.
Overflow of urine—from paralysis of the bladder.

CAUSES OF DEATH.

Death may occur from bed-sores, cystitis (from stagnation of urine due to paralysis); tuberculosis, or some intercurrent disease; pyelitis; ammonæmia.

PROGNOSIS.

May run on for years and then remain stationary. Recovery is rare. Gradual advance to death from complications.

TREATMENT.

Is of very little service. The actual cautery and hot iron should be applied near the supposed seat of inflammation. A sponge dipped in hot water may be placed along the spine, especially where the inflammation is most severe, with some benefit.

LOCOMOTOR ATAXIA.

SYNONYMS.

"Tabes dorsualis,"—or gray degeneration, or softening of the posterior columns of the cord.

DEFINITION.

Is a disease characterized during life by imperfect co-ordination of muscular movement and sensory manifestations; and after death by disease of the posterior columns of the cord.

MORBID ANATOMY.

The disease is confined to the posterior columns of the cord; the dura mater is thickened and opaque. The arachnoid is moderately opaque, and there is fluid in the sub-arachnoidean space.

The pia mater is thickened, opaque, and adherent over the affected part. The posterior columns are degenerated, the degenerated portion is wedge-shaped, with its apex directed toward the centre of the cord. It appears as a reddish, soft mass. The posterior roots of the spinal nerves are degenerated and the antero-posterior diameter of the cord may be shortened, due to degeneration and possibly sclerosis.

The *microscope* shows few nerve filaments in various stages of atrophy, and, between the cells, nucleated connective-tissue from proliferation.

ETIOLOGY.

It may follow exposure to dampness, as in the case of fishermen; abuse of alcohol; syphilis; masturbation; excess of venery; severe muscular strain, as in long marches, etc.

SYMPTOMS.

Premonitory symptoms.

Tearing localized pains in the lower half of the trunk which are more or less paroxysmal (may last for a long time, even for years, before ataxia occur).

Delayed sensation and *absence* of "*patellar reflex.*"

Sense of formication in the lower limbs.

Sense of numbness in the lower limbs.

Sensation as if a ligature were tied around the body.

If the disease be high up.

The upper extremities may be affected as well as the lower, although the lower limbs are chiefly involved.

Sense of insecurity in walking—especially when any sudden movement is required, as in crossing a street.

Abnormal sensation in walking—as if walking on air or wool.

No dragging of the limbs exists—but *the limbs are forcibly lifted* and thrown in the wrong direction.

The patient is liable to fall if the eyes be closed—or if walking in the dark (this must not be considered pathognomonic of this disease).

No motor paralysis, or atrophy in any special muscles—as you can move and put the limb in any position and retain it there in spite of a counter-extending force.

Sexual intercourse is often decreased, but priapism may be present.

In the advanced stage.

Walking—is almost impossible; the feet are swung in a circle, the heel striking the ground first, and then the sole of the foot; the gait is jerky in character; the patient may fall from inability of co-ordination of the muscles, and often loses his equilibrium.

If the upper part of the cord be affected, the patient cannot button his coat or collar, neither can he carry a glass of water steadily to his mouth (since co-ordination of movement is demanded) and the handwriting is markedly affected.

Paralysis of the sphincter vesicæ (incomplete enuresis, due to too long retention).

Diplopia
Strabismus } from paralysis of the third and sixth nerves.
Ptosis

Pupil fails to respond to light, but alters its size in efforts of accommodation of vision.

Cerebral disturbances.

CAUSES OF DEATH.

Death may occur from bed-sores (from enuresis); severe cystitis; pulmonary consumption; and intercurrent diseases.

PROGNOSIS.

Recovery is extremely rare.

TREATMENT.

If there be a suspicion of syphilis, use the anti-syphilitic remedies. If from having taken cold, *leeches* and *blisters* should be applied along the spine; sweating in moist clothes may be of benefit; and the continuous current along the spine and the course

of the spinal nerves may possibly prevent the extension of the disease.

PROGRESSIVE MUSCULAR ATROPHY.

MORBID ANATOMY.

The muscles are decreased in size, pale, and yellow, with bundles of atrophied and degenerated tissue; and in the later stages, degeneration of the whole muscles.

The microscope shows paleness of the muscular fibrillæ; no transverse striæ, fine granular fat-globules in the centre of the fibrillæ; later on, collapse of the sarcolemma and fat-globules inclosed. The muscles of the heart, intestines, or bladder are never affected. It affects principally the muscles of the hand, hip, or shoulder. It may affect one muscle, or group of muscles.

ETIOLOGY.

It is a primary disease of the muscles, and degeneration of the anterior roots of the spinal nerves, due to disease of some small part of the brain, or spinal marrow, and over-work of the muscles. It attacks all ages and both sexes.

SYMPTOMS.

Increased feeling of weakness in certain muscles or group of muscles.

Rapid disappearance of muscles—commencing in the hand, hip, or shoulder, causing alteration in the ball of the thumb, flattening of the shoulder, and prominence of the spinous processes of the vertebræ.

Loss of muscular action of the affected side.

A peculiar fibrillar twitching (as though the skin were alive) in the weak and atrophied muscles, but no movement of the joints.

A loss of sensory muscular nerve power.

The electric current does not fail to induce contraction until the muscle is entirely atrophied (pathognomonic).

The thigh may become even smaller than the calf of the leg when the muscles are greatly atrophied.

Intellect is clear throughout.

In advanced cases.

Patient cannot walk or change position.

Patient requires to be fed.

Arms hang loosely to the sides of the body.

Loss of expression of the countenance } evidences of extension of spinal changes upward.
Saliva flows from the mouth }

Speech is indistinct (due to extension of disease to medulla).

Tongue cannot pass the food into the pharynx, even after it is placed in the mouth.

CAUSES OF DEATH.

Death may occur from atrophy of the muscles of respiration, and deglutition, causing failure in the performance of their function.

TREATMENT.

The induced or constant current should be frequently applied with great patience and perseverance.

SUNSTROKE.

SYNONYMS.

Coup de soleil; Calenture; Insolation; Sun fever; Heat apoplexy; Œgyptian starstroke; Heatstroke; Thermic or heat fever.

DEFINITION.

Is a disease characterized by a more or less sudden attack of unconsciousness due to exposure to high temperature under adverse circumstances.

MORBID ANATOMY.

In most cases of fatal sunstroke the blood is found to have remained uncoagulated; the lungs intensely congested; the right side of the heart loaded with blood; and generally more or less

engorgement of the vessels of the brain. The body also loses its heat very slowly after death. Some observers assert that a slight increase usually takes place.

ETIOLOGY.

Exposure to an intense heat where the atmosphere is damp and impure, and the patient exhausted by over-exertion or from any means which impede the free action of the respiratory muscles. In tropical climates it frequently occurs from exposure to the direct sun rays and want of acclimatization. It is also very common at night-time among those subjected to close, over-crowded, ill-ventilated barracks, tenement houses, ships, etc; soldiers exposed to long marches in the heat of the day. Intemperate and debilitated persons and those who drink largely of iced-cold water when greatly exhausted are very liable to be affected.

SYMPTOMS.

Onset may be sudden but frequently preceded by premonitory symptoms.

When onset is sudden.

Patient falls unconscious.

Head—intensely hot.

Skin—pale, cold and moist.

Respiration—gasping and is apt to be stertorous.

Pulse—extremely feeble and rapid.

Temporal arteries—greatly distended.

Temperature internal—above normal.

Convulsion may precede death which takes place very rapidly or even quite suddenly.

When onset is gradual.

General malaise—which may be present for several days.

Skin—dry and hot.

Nausea.

Frequent desire to micturate.

Restlessness or *insomnia*.

Face—flushed.

Eyes—congested.

Constipation.

Vertigo.
Headache—intense.
Vision becomes dim.
Weakness in the limbs and *great prostration.*
Coma—which may be partial or complete, when
Breathing—stertorous.
Pupils—contracted.
Face—pallid.
Skin—intensely hot.
Temperature—varies from 100 to even 109°.
Pulse—rapid and full and as death approaches becomes weak, irregular but very rapid.
Convulsions—sometimes.
Bowels—relaxed.
Vomiting—not infrequent.

SEQUELÆ.

Headache; chorea-like affections of the muscles; epilepsy; and some degree of mental imbecility; amaurosis.

DIFFERENTIAL DIAGNOSIS.

From apoplexy; delirium tremens; insaniiy; eclampsia; paralysis.

PROGNOSIS.

Is unfavorable, the mortality being very heavy, exceeding, it is said, forty per cent.

TREATMENT,

Depletion should never be resorted to. The patient should be placed in a cool room; his clothes removed and a *cold shower bath* administered. The shower being at least from four to five feet above the patients head. When removed from the bath the head should be shaven and cold applied to the head. If there be dyspnœa *dry cups* placed on the thorax in front and behind will be of service. *Bromide of potassium* gr. v.–xxx. should be given in all stages of the affection and in mild cases (gr. v.–x.) may be administered every half an hour to an hour until the grave symptoms disappear; if the patient be unable to swallow it can be

given by injection remembering that the dose should always be increased one quarter more than when administered by the mouth. Stimulants such as brandy and milk, or brandy and ammonia, should be given whenever exhaustion is prominent and the cold douche dispensed with whenever the skin is cold. After return of consciousness, blisters or sinapisms should be applied to the back of the neck or behind the ears. The bromide should be continued for at least two weeks after the return to consciousness. When convenient, the patient should be sent to a cool district and free from all excitement or mental or physical exertion. Regular habits and exercise should be enforced. In order to prevent or lessen the danger from nervous affections which are apt to follow sunstroke, a continuance of this treatment for some months should be adhered to.

COMA—ITS PRINCIPAL CAUSES AND DIFFERENTIAL DIAGNOSIS.

The most frequent causes of coma, (see page 26), comprise, syncope, cerebral concussion and compression, asphyxia, epilepsy, apoplexy, embolism, hysteria, catalepsy, uræmia, alcohol, and opium.

Syncope.

Pulse, feeble; face, pale; respiration, quiet; duration, short.

Cerebral Concussion.

Pulse, rapid and feeble, and intermittent; skin, pale and cold; respiration, feeble and sighing; pupils, usually contracted; coma, incomplete, is usually immediate, and rapidly decreases, as a rule; temperature, lowered; eyelids, usually open and movable; vomiting, frequently present; incontinence of urine and involuntary evacuations occur; duration, short.

Cerebral Compression.

Pulse, full and slow; skin, usually warm and moist; respiration, slow and stertorous; pupils, may be natural, dilated, or irregular;

coma, profound, may not directly follow the injury, and is stationary or increasing; temperature, normal or increased; eyelids, closed and immovable; vomiting, rare; retention of urine and obstinate constipation; paralysis (modified by seat of lesion).

Asphyxia.

Face, livid; lips, blue; extremeties, blue and cold; respiration, distressed.

Epilepsy.

Epileptic cry on onset; tongue, ragged; history of previous attacks; pupils, dilated; mouth, bloody froth exudes; spasms. tonic and clonic; body, one side more convulsed than the other, reflex sensibility present; duration, short, and followed by a deep sleep; loss of consciousness of short duration.

Apoplexy.

Coma, may occur at onset or develop rapidly, and deepens; hemiplegia; pulse, slow and full; face, flushed; pupils, irregular; respiration, stertorous; condition of arteries, usually atheroma of those which are superficial; age, usually occurs at about 50 years; history, high living, etc.; muscles, rigidity or relaxation.

Embolism.

Advent, is sudden, with consciousness; paralysis, usually of the right side; improvement, is usually marked within twenty-four hours (due to collateral circulation); face, pale or normal; respiration, sighing; *aphasia*, usually present either of the amnesic or ataxic variety; age, may occur at any time; pupils, dilated or irregular; history, may be rheumatic, or evidence of mitral or aortic disease may be detected on auscultation.

Hysteria.

Globus hystericus; large amount of pale urine in the bladder, which is passed after the attack; a previous history of attacks of hysteria; most common in females.

Catalepsy.

Limbs, at first rigid, then become loose and will remain in any position in which they may be placed.

Uræmia.

Is of gradual onset; face, pale and waxy; pupils, dilated; eyes, closed; coma, complete; œdema, of face and limbs; uræmic odor of the body, and especially of the breath; urine, contains casts and albumen; history of convulsions.

Alcohol.

Pulse, rapid; face, flushed; skin, warm and moist; respiration, stertorous and puffy; pupils, dilated, and respond to light; no paralysis; breath, alcoholic; vomiting, occasional; eyes, injected; temperature, below normal; feet, are drawn back when tickled; history of drinking.

Opium.

Onset is gradual; face, dusky; skin, moist; pulse, slow and full; pupils contracted, (pinhole); respiration, slow and irregular, and there may be an interval between expiration and inspiration; patient can be roused till coma deepens; vomiting (the matters vomited have the odor of opium); breath, has the odor of opium; the attack tends to deepen, the respiration gradually becoming slower and slower as the drug exerts its influence. The patient, when roused, answers questions rationally, in marked contrast to some other forms of coma (especially alcoholism).

DISEASES OF THE BLOOD.

ETIOLOGICAL DIVISION OF FEVERS.

1. *Miasmatic contagious.*
   The poison must be deposited in decomposing organic matter exterior to the body, so that, when once in a body or system, whose condition is impaired or weakened, the poison is reproduced. It can only be conveyed through the excrements of the sick, or through decomposing organic matter exterior to the body, with which such excrements have come in contact. They may be endemic or sporadic (as in typhoid and yellow fever).
2. *Malarial or miasmatic non-contagious.*
   Where the morbific agent is developed exterior to the body (as in simple intermittent, simple remittent, pernicious, dengue, and typho-malarial fevers).
3. *Contagious.*
   Where the morbific agent is developed in the body, and can be transferred through the atmosphere to another (as in typhus, measles, small-pox, relapsing, scarlet, and miliary fevers.

TYPHOID FEVER.

DEFINITION.

Is one of the miasmatic-contagious fevers.

SYNONYMS.

Autumnal, abdominal-typhus, enteric, gastric, gastro-enteric, forty day, dothenenteria, ilio-typhus, typhoid affection of Louis.

MORBID ANATOMY.

There is the change in the blood which becomes darker in color, imperfectly coagulable, and the serum is of an unnaturally yellow color. Through these blood changes, the various tissues and

organs in which the process of repair and waste are carried on become affected, as the spleen, liver, kidneys, heart, lungs, bronchial tubes, larynx, brain, stomach, muscles, and salivary glands.

Spleen.—Is sometimes *enormously increased* in size (for about three weeks), becoming soft, of a dark jelly-like mass, easily broken down (from congestion); or there may be infarctions and rupture, but if recovery take place, the spleen will return to its normal size.

Liver.—May be normal, or soft and flabby, with more or less granular, fatty, and lymphoid cells; or there may be catarrhal or diphtheritic inflammation, or ulceration of the lining membrane of the gall-bladder.

Kidneys.—*Degeneration* may take place, especially in the epithelial elements of the cortical substance, but not so markedly as in typhus.

Heart.—*Parenchymatous changes* are more marked than in any other organ (from faulty nutrition); becomes soft and flabby, and of grayish or brown color; is easily broken down, and the walls of its cavities readily fall together (on account of granular degeneration); its muscular fibres have an amyloid appearance, which does not answer to the iodine test (this change also takes place in the voluntary muscles of the body). There may be thrombi in the heart, or vegetations on the valves, or chordæ tendineæ, producing infarctions and feeble heart-sounds, and the first sound may be entirely absent.

Lungs.—*Undergo splenization;* they may be of a dark reddish-blue, black, or brown color, harder and drier than normal, and when cut, a dark watery fluid oozes out (due to stasis in the capillary circulation).

Death may ensue from pulmonary infarctions. These infarctions in the recent state are dark and feel like consolidated lung-tissue, but later may change to a yellow color, or may soften and break down.

Bronchial tubes.—There is *extensive catarrhal inflammation* of the larger tubes, generally giving rise to capillary bronchitis or broncho-pneumonia.

Larynx.—There may be *extensive catarrhal inflammation.*

Ulcers (called typhoid ulcers) may occur, causing extensive hemorrhage (this type of ulceration may affect the mouth and pharynx, or even the epiglottis or mucous membrane of the Eustachian tube, producing deafness).

Brain and nervous system.—*Œdema of pia mater and brain-substance* may exist, with extensive adhesions to dura mater (not much, however, is known of these lesions).

Stomach.—Changes in this organ are sometimes extensive. Softening and degeneration of its glandular structure occur, which are the causes of disturbances in digestion during the fever and even during convalescence.

Muscles (voluntary).—There may be granular or fatty degeneration; or a waxy or amyloid degeneration. The muscular fibres, in both instances, become thick and friable, and are yellowish-white in appearance. The want of muscular power of fevers depends on the nervous disturbance, but the excessive loss of muscular power in convalescence is due to muscular changes.

Salivary glands.—These become enlarged, tense, and firm; are of brownish-yellow color, and later on they become red (due, first, to cellular hyperplasia, and secondly, to degeneration).

Intestinal lesions are *the most important* of all the lesions, and are characteristic. The poison acts on the mucous membrane of the small intestine, producing catarrhal inflammation and changes in its structure. These changes are modified by the duration of the fever, and their nearness to the ileo-cœcal valve. They are *most marked in the patches nearest the valve* (ileo-cœcal).

The intestinal changes in the regular course of a severe case may be thus summarized:

First week.—The mucous membrane around *Peyer's patches* becomes *congested* and *swollen;* the glands become more and more elevated; and the surfaces are of a dark-red color, interlaced by white lines ("shaven beard appearance"). These changes are most marked nearest the ileo-cœcal valve.

Second week.—The mucous membrane of the intestines becomes less red; the solitary glands more elevated; the white lines on their surface disappear; and are uniformly red. There is an *excessive cell growth in the follicles* (due to rapid development of

lymphoid cells, principally within their structure, causing pressure on the capillaries and interfering with the circulation of gland-tissue). Toward the end of the second week, degenerative changes are established in the new tissue, which may become disintegrated, absorbed, and undergo resolution; or the follicles may rupture into the intestines, or more frequently the dead tissues slough and ulcers are formed.

Third week.—These *ulcers* are yellowish on account of staining by bile. When sloughing takes place, the entire gland and mucous membrane surrounding it are removed and the muscular coat laid bare. The edges of the ulcers are sharp, swollen, and overhang the floor of the ulcers, and *may terminate in perforation of serous coat and fatal peritonitis.* These ulcers may be hemorrhagic, and destroy life in this manner.

Fourth week.—Cicatrization commences. The swollen edges subside. Granulations spring up from the base of the ulcer (which is the muscular coat of the intestine); connective-tissue is formed; edges unite at their base, and are covered with a layer of epithelium. The cicatrix is depressed, becoming more and more pigmented, but seldom causes any puckering or diminution in the calibre of the intestines. *Death* may result *from prolonged ulceration.*

Mesenteric glands.—Those nearest the ileo-cœcal valve are affected secondarily to the intestinal glands. They are first congested, then lymphoid and large cells are produced, causing enlargement and acute cellular hyperplasia; and, about the end of the second week, the glands become disintegrated. They are often about the size of a walnut or hen's egg. Some of the glands shrink to their normal size, others soften and are absorbed, and leave a fibrous cicatrix. When of very large size, absorption may be incomplete, and a dry, yellow cheesy mass is left; salts of lime are often deposited in them, and they become inclosed in a fibrous capsule; or they may become fluid, their capsules destroyed, enter the peritoneal cavity, and produce peritonitis.

Suppurative inflammation in the cellular-tissue, on the surface of the body, may be another lesion occurring during convalescence, accompanied by redness and pain. A tumor of a fluctu-

ating nature is formed wherever there is the greatest pressure, or two or more of them may coalesce.

Bed-sores (in later stages) occur from imperfect skin nutrition and prolonged high temperature. They are most frequent over the trochanters or sacrum, or wherever there is pressure, and may involve the muscular structures.

Gangrene of the toes may occur, due to thrombosis or embolus, *from pressure* on circulation of lower extremity.

SYNOPSIS OF THE PATHOLOGICAL CHANGES.

The *pathognomonic lesion* is confined to *Peyer's patches* and *mesenteric glands.* In these, the following changes occur:

1st. *Congestion* of Peyer's patches (producing "shaven beard" appearance).

2d. *Hyperplasia* and *hardening* of these glands.

3d. *Softening of glands* from fatty degeneration.

4th. *Absorption or rupture*, with evacuation of contents. In case of rupture, an ulcer is left, whose long axis is parallel to the length of the intestine; the edges are ragged and undermined, and the floor is formed by the muscular wall of the intestine. The ulcers are on the unattached border of the intestine.

5th. *Cicatrization* of these ulcers, and a pigmented cicatrix. In severe cases, perforation at the seat of ulceration may occur. The mesenteric glands undergo enlargement, and softening through fatty degeneration.

All of the organs are generally softened and fatty, and the muscles are in a state of fatty degeneration. There is extreme emaciation, and the common lesions of all fevers exist (see page 316).

ETIOLOGY.

The poison, which is contained in the fæcal discharges of the sick, if fresh, undergoes development outside the body, especially in soil saturated with organic matter, and gains access to the system by the air we breathe and the water we drink. The period of incubation is from fourteen to twenty-one days. It is more frequent in the autumn than any other time of the year. It oc-

curs more frequently between the ages of 15 to 25. Exposure, bad nutrition, and debility of system favor its development.

SYMPTOMS.

Early symptoms of general malaise.

Vomiting of greenish fluid.
Headache (lasts from seventh to twelfth day).
Intolerance to light.
Pains in limbs (all over the body).
Chills and fever, but not well marked.
Slight diarrhœa.
Variations in temperature.

Temperature—

First week.—Gradual and steady rise morning and evening. In mild cases 103°. In severe cases 105° to 106°. High in morning (from 12 to 6 P.M.). Low in evening (from 6 P.M. to 8 A.M.). *Pulse* 98 to 110, and increases about five beats for every degree of temperature.

Second Week.—Temparature and pulse remain at their maximum till the end of this week when a decline usually occurs; may be rapid and feeble. *Pulse* may go to 120 to 140. If 140, it denotes feebleness of heart's power, and is a *bad omen* if it remains so for five or six days.

Third week.—Variations are remittent in character. If pulse and temperature keep high, is fatal. *Heart-sounds may be inaudible,* and prompt attention to the patient is then indicated.

Fourth week.—Variations are intermittent and exacerbations not so severe.

Eruption—begins from sixth to twelfth day, and remains till the fourteenth day; consists of lenticular spots scattered over abdomen and chest, are *of a bright rose-color, and disappear on the slightest pressure and return directly. Each spot remains three days and then disappears, when another batch takes its place.* The latter is the distinguishing point of the eruption. Da Costa says it resembles flea bites.

Delirium—low muttering, and often becomes maniacal at night.

Diarrhœa—if slight throughout, is not a bad sign, but excessive diarrhœa is an unfavorable symptom. Generally commences about second week, is alkaline, and of a yellow-green color (*pea soup*).

Stupor.—This is a common symptom.

Great emaciation is marked, if the disease be typical.

Subsultus or twitchings of the wrists, picking at the bedclothes, may be present.

Involuntary evacuations and micturition.

Tongue—is at first of a light white color, then red on the tip and sides, and dry in the centre; then *heavily coated, with sordes on teeth and sides of the mouth.* May become clean and shining like raw beef, or the tongue and lips may be *dry and cracked,* and the mouth covered with dark incrustations. Hemorrhage from the gums may also occur.

Countenance – pale, olive, or leaden look. Eyes dull and pupils dilated. Conjunctiva congested, hectic flush, and a sunken and anxious expression.

Tympanites—(from gas in the intestines, due to changes in mucous membrane), indicates danger, if excessive.

Pain and tenderness in right iliac fossa—about the sixth day (*never press with the tips of the fingers lest the intestine be ruptured*); pain may be due to perforation, when there will first be sudden diarrhœa, and aggravated pain on pressure in the abdomen; the pain of typhoid may be located, at first, in right iliac fossa, but soon extends over entire abdominal cavity.

Intestinal hemorrhage—from a trace to 16 or 18 ounces, is preceded by sudden rise in temperature and extreme fall. If profuse, it is due to the opening of an artery with an intestinal ulcer, and preceded by fall of temperature, etc.

Nausea—(later on in the disease).

Vomiting—(later on in the disease) which may be due to acute gastric catarrh or general peritonitis.

Cyanosis and collapse—(later on in the disease).

Epistaxis—may occur during the first week and is of little importance, but when it occurs during the third week it may be so profuse as to destroy the life of the patient.

Somnolence.
Muscular prostration and, in severe cases, paralysis may develop.
Aphonia.
Deafness — due to ulceration of mucous membrane lining the Eustachian tube.
Perverted taste.
Excessive hyperæsthesia.
Enlarged spleen.
Urine—is first diminished and then increased. Albumen very rare.
Lungs—typhoid râle, a sonorous, dry, ringing sound, is often present and is due to a bronchial complication.

If convalescence take place, the edges of the tongue will be moist, this moisture extending all over the tongue.

SYNOPSIS OF THE SYMPTOMS.

First Week.—Temperature, mild cases, 103°. Severe cases, 105° to 106°. Pulse, 98 to 110. Epistaxis; slight diarrhœa; tympanites; and tenderness over right iliac fossa and gurgling (bad omen).

Second Week.—Temperature and pulse may fall to normal, or pulse go up to 120 to 140 (about the seventh day). *Eruption* (from sixth to eighth day) generally commences at this time and remains till the fourteenth day; appears on abdomen and lower part of the chest, may be scanty and is of a *bright rose color*, etc. Marked diarrhœa (resembling *pea soup*). Delirium—low muttering, or maniacal. Coma vigil. Subsultus tendinum. Picking of the bed-clothes. Symptoms of bronchitis; tongue brown, dark, and incrusted; sordes on teeth, etc.

Third Week.—Epistaxis, or intestinal hemorrhage (alarming), and other fatal symptoms. If convalescence take place, the eruption disappears gradually, as do also the other symptoms. *There is, in the convalescent stage, danger of perforation for six months.*

DIFFERENTIAL DIAGNOSIS.

In early stages, with typhus, relapsing, typho-malaria, acute tuberculosis, pyæmia, septicæmia, pneumonia, gastro-enteritis, or trichonosis.

Acute Tuberculosis.

Temperature—106° or 107°, (and no remissions).
Spleen—not enlarged.
No eruption.

Typhoid Fever.—Temperature, rarely 106 (with remissions).

Enlarged spleen.—Eruption, red-rose color.

In trichonosis.

No eruption ; pain in the joints (resembling acute articular rheumatism); and by taking a piece of the pectoral muscle and placing it under the microscope the parasite will be seen, which determines the diagnosis.

In Pyœmia and Septicœmia.

Jaundiced hue to surface; no eruption; early symptoms of profuse sweating; emaciation; delirium, etc.; history.

In Typhoid Pneumonia.

Typhoid symptoms; coma in later stages; cough and expectoration; no variation in temperature.

In Gastro-enteritis.

Diarrhœa and vomiting precede the fever, and the temperature is never higher than 103°.

PROGNOSIS.

So long as abdominal symptoms last, the patient is not out of danger, as changes are still going on. If the temperature be not higher than 104° to 105° on the eighth day, and is regular in its development—the prognosis is favorable.

In all cases, the heart is the guide; for, if pulse be 110 to 115, and there be a distinct first sound; and, at the end of second week, the temperature be even 106°—favorable. If pulse be 120 to 130, feeble apex-beat, first sound indistinct or obscure, and tendency to cyanosis—be on your guard.

It is the rise and fall in pulse that denotes the danger. The prognosis is always bad, in fat persons and gouty subjects, or if complicated by kidney troubles. There is great danger of peritonitis from ulceration and hemorrhage, if the attack be severe. Bad prognosis, in alcoholists and when occurring in connection with chronic diseases. Bad, in complications, as diminished heart's action from muscular degeneration, favoring pulmonary and other hypostatic congestions. Bad, in intestinal perforations, capillary bronchitis, œdema of the lungs, latent pneumonia, laryngitis (from protracted fever and prostration, causing ulceration of mucous membrane) pyæmia, acute gastric catarrh. Bad, if brain symptoms occur, as œdema, hemorrhagic extravasations, hemiplegia,

and paraplegia, also in parenchymatous nephritis, severe catarrh of bladder, cellulitis, and bed-sores.

Death may result from toxæmia, asthenia (debility), suppression of excretory functions of kidney, hyperæmia, and œdema of the lungs, intestinal hemorrhage, exhaustive diarrhœa, intestinal perforation, peritonitis, with or without intestinal perforations; relapse from reabsorption; and from cathartics, if given during first week.

TREATMENT.

Always use a porcelain bed-pan, covered over the bottom with a layer of powdered sulphate of iron, and, directly after the discharge, cover it all over with muriatic acid (about equal to one-third of the mass). *Dig trenches*, but not near a privy or drain, and empty contents of bed-pan into these trenches. *Remove all soiled bed-clothes, immerse them in chlorine water*, and boil them within twenty-four hours. Examine the pipes, wells, etc., etc., and see the house sewer-pipes do not leak. Disinfect thoroughly, for after the poison has once entered the system there is no preventive.

Arrangements of the sick-room are of the utmost importance. *An experienced nurse. Large well-ventilated room. Remove all carpets* and all superfluous things. *Temperature of the room* 60°. No fruit or solid food. Patient must be kept in bed. *Milk*, ad libitum. In early stage (temperature 103°), sponge surface with cold or tepid water. Keep down temperature with *quinine* and *cold* (using bath, packs, and affusions). If temperature rise above 103° in axilla, then warm bath, 70° to 80°, and gradually lower the temperature of the bath by ice or water till patient begins to feel the effects; probably it will have to go to 60°, but not below this. If temperature fall rapidly to 103°, remove patient at once; but, if slowly, do not remove patient till temperature of 101° be reached, and apply cold to head; this should only be done during first and second week. *Cold pack* should be thus applied: wring out a sheet in tepid water, and one in cold water, and wrap round the patient; or apply ice-bags to abdomen. Be very careful when using cold applications.

Quinine, in large doses, 30 *to* 40 *grains within two hours, or* 10

grains every half-hour for four doses, but do not continue it when once temperature is reduced, unless it be 105°, then reduce to 101°, and keep it there. If, after the third week, you fail to reduce the temperature, *add gr. x.-xx. pulv. digitalis*, unless pulse be frequent and irregular, then only use quinine.

If weakness and failure of heart's power occur during the third week, and the temperature be about 101°, pulse 140, with first sound inaudible, subsultus, dry tongue, etc., *use stimulants, but be very careful of the first few doses, and see your patient every two hours*, so that, if he be restless, delirium more active, temperature higher, pulse more and more rapid, *stop it at once;* but, if pulse become fuller and more regular, heart-sounds more distinct, less delirium, and restlessness, then continue it.

Nutrition.—No beef-tea—cannot digest it, and little nourishment in it; give *milk and lime-water*, which is an antiseptic.

Diarrhœa denotes intestinal changes from inflammation and subsequent intestinal catarrh. In early stages, leave it alone for first or second week. *If it commence late* (during latter part of third or fourth week), it is due to ulceration or sloughing. It *should be arrested or held in check by opium hypodermically, in small doses*, and not by astringents. Stimulants can be administered at this stage.

Tympanites.—Use *turpentine stupes to abdomen*. Dip a piece of flannel in boiling water, wring it out, pour turpentine over it, and apply to abdomen.

Intestinal hemorrhage may cause death from exhaustion. If occurring late, should be promptly arrested. Patient must be *kept in bed, and semi-narcotized with opium, given in small doses* at intervals; must remain perfectly still in bed. If hemorrhage be profuse, patient must be kept under the influence of opium for a week or ten days.

Peritonitis.—Death occurs as a result of general peritonitis from perforation in twenty-four hours. *If without perforation, then semi-narcotize your patient for seven to ten days.* Be careful of cathartics and enemata; do not use them, even if no passage occurs for two or three weeks. If stomach be irritable, use *hypodermic injections of morphia* over abdomen, sufficiently large to check peristalsis.

Bronchitis.—If capillary bronchitis, *apply dry cups* to chest, and ammon. carb. internally.

Pneumonia.—*Stimulants* should be given, or increased, if previously used, at the time pneumonia occurs.

Laryngitis.—Small blister below the angle of the jaw, and a poultice round the whole neck. If suffocation seem likely to occur, then perform tracheotomy.

Subacute gastric catarrh.—*Rest the stomach;* give ʒ i. milk at a time, and hot fomentations over epigastrium.

Bed-sores.—Scrupulous cleanliness and bathe the parts in spirits of camphor, and relieve the points of attack from pressure. If the sores penetrate the soft tissues, then wash frequently with carbolic acid, or brush the part over with equal parts of *balsam of Peru and copaiba,* and cover with dry lint and vaseline. *If gangrenous,* use continuous warm bath, and so soon as sloughing takes place and parts separate, dress the parts with lint saturated in balsam of Peru and carbolic acid.

Delirium.—*Chloral, gr. x.-xx.*, and perhaps repeat in two hours; or give *stimulants,* if associated with insomnia, and especially if there be anæmia.

Jactitations, etc.—*Inject hypodermics of sulph. ether* (ʒ i.) in four different parts of the body.

Convalescence.—Eat often and little, such as milk, cream, jellies, gruel, animal broths. *No solid food;* and, if diarrhœa be present, *only* milk and cream. No exercise, except walking about in sick-room. Patient should be kept in semi-recumbent position for two or three weeks after convalescence. If convalescence be slow, give small doses of *quinine, iron,* and *oleum morrhuæ* after food. If diarrhœa occur, keep patient in bed, give catechu and hæmatoxylin, and, if convalescence be delayed, then send him away for change of air.

YELLOW FEVER.

DEFINITION.

Is a miasmatic-contagious, and also an infectious disease.

MORBID ANATOMY.

The characteristic lesions, after death, will be found in the liver, kidneys, and the blood.

Liver—is sometimes the color of fresh butter, mustard, coffee and milk, or chocolate. This change may be confined to one lobe or portion of the lobe, or it may extend over the whole liver. The organ contains less blood than normal, and is dry in appearance, softer than normal, and breaks down easily upon pressure.

The *microscope* shows the hepatic cells infiltrated with oil globules, or there may be a granular change and disappearance of the cell-nuclei. This change is called "acute fatty degeneration."

Stomach.—The mucous membrane is thick, red, and soft.

Heart.—Is soft, flabby, lighter in color than normal, and the outline of the fibres is lost (from a degeneration which is taking place).

Lungs.—Are the seat of hemorrhagic infarctions.

Kidneys.—Are increased in size, due to an increase in the cortical substance. It is a true parenchymatous nephritis, in which the fatty stage is rapidly reached. The *microscope* shows the uriniferous tubules crowded with oil-globules; in some places they are denuded of the epithelium, and in others filled with broken-down epithelium, which is undergoing a fatty and granular change.

Brain.—Will be found hyperæmic.

Spleen.—Is darker, softer, and slightly enlarged, if at all.

Skin.—Varies from a bright yellow to a dark orange-color.

Blood.—Is changed in color; darker than in health; the globules have not the usual rounded outline; their edges are serrated; they break down easily, and rapidly undergo ammoniacal changes. The blood coagulates less rapidly and less perfectly than normal (this is due to a diminution or loss of the coagulating power of its fibrin).

Other organs.—There is acute catarrh of the mucous membrane of the intestinal tract and larynx. The veins become turgid from intense hyperæmia often resembling varicosities.

ETIOLOGY.

This disease is rarely met with north of 40° north latitude, or

south of 20° south latitude, and for its production it is necessary to have decomposing animal or vegetable matter, a certain amount of moisture, and a temperature which must be above 77°, together with the specific poison. When the southerly winds prevail, the epidemic spreads and increases in severity, and when the northwest winds prevail, it is arrested, and its ravages are stopped so soon as the frost sets in. It is a portable disease, and occurs in sea-port towns. Its period of incubation varies from twelve hours to four or five days, or even several weeks.

STAGES.

Invasion—Remission—Exacerbation.

SYMPTOMS.

Ushering in Symptoms.

General malaise—which occurs for two or three days previous to the attack.

In mild cases.

Chill—with or without rigors.

Fever—continues from a few hours to two or three days.

Temperature—varies from 102 to 110° F.: reaches its maximum at the end of second day (which is rarely higher than 105°).

Pulse—seldom above 100.

Tongue—moist and somewhat coated.

Headache—supra-orbital and intense.

Vomiting—which is not prominent till late in the disease.

Tenderness on pressure over the epigastrium.

Constipation—which may be intense.

Delirium—rarely violent.

Pain—in the lower limbs *especially in the calves of the legs.*

Eyes—red, irritable, watery or tearful.

Fever abates or ceases after two or three days when a remission occurs, commonly termed the state of calm.

In severe cases.

First stage.

Pains, etc., may cease.

Temperature—rises rapidly, may reach as high as 110°.

Pulse—seldom higher than 110—termed the "gaseous pulse." Is easily compressed and uncertain in character and volume. It may fall below normal (even to 30 or 40), is small, weak and vibratory.

Lividity of back—due to hypostatic congestion.

Vomiting—projectile or *regurgitant*, a most constant and *characteristic* symptom. It comes on after the chill, continuing throughout the whole course of the fever; at first it consists of the contents of the stomach, and is alkaline, then of a thin liquid which may be of a red, claret, or brown color. Finally the *black vomit* appears, which may occur on the second or third day (but usually ceases from twelve to twenty-four hours before death); consists of pigment (produced by changes in the blood by the gastric juice) and globules (resembling blood); also epithelial cells from the mucous membrane of the stomach, degenerated lymphoid cells and white blood globules. It is acid and has an acid taste without bitterness; it may be acrid and excoriate the throat, tongue and lips.

Tenderness over the epigastrium—which may be extensive.

Tongue—usually red, dry and cracked but may be moist and covered with a creamy coating.

Second stage (*stage of remission*—may last from a few hours to two or three days).

Temperature—by the fourth day falls very rapidly, but usually not below 100°. It again rises quickly to 104°, remains stationary for 24 or 48 hours then falls to normal till convalescence is established.

Evacuations—brownish or nearly black (due to presence of blood passing through the intestinal canal).

Diarrhœa—rare.

Albuminuria—frequent in grave and fatal cases.

Urine—usually scanty and contains hyaline and granular casts, red blood corpuscles and renal and vesical epithelium. It is acid in the early stage and afterward alkaline due to the presence of bile when it may be of an orange color (from bile staining and blood). Previous to a fatal termination there is always complete suppression.

Surface of the body—yellow (occurs after the paroxysm of fever).

Eyes—red (like a ball of fire) and watery, and have a brilliant glassy transparent look.

Jaundice—hepatogeneous (usually in fatal cases).

Hemorrhages may occur from the stomach, intestines, kidneys, bladder, uterus, nostrils, gums, etc.

Petechiæ and vibices—sometimes observed.

Coma and convulsions—may occur (due to uræmia).

Delirium—may occur (usually during the fever).

Muscular prostration.

Facies—suffering; dejection; anxiety; anguish; despair; terror; stupidity; vacancy, astonishment; sullen; etc.

Third stage—(stage of collapse or exhaustion).

Prostration and feebleness.—Pulse, irregular.

Extremities, cold.—Delirium, low.

Indifference and apathy (as in epidemic cholera) showing tendency to death by asthenia.

Restlessness—jactitations and great distress may occur.

Death or convalescence.

If convalescence occurs it is slow and preceded by fever with typhoid symptoms.

DIFFERENTIAL DIAGNOSIS.

From malarial fever; relapsing fever; bilious remittent; and acute yellow atrophy of the liver.

PROGNOSIS.

Is unfavorable. The duration of the fever is usually about six days. It often destroys life in three days. The average ratio of mortality is that, in every three cases during an epidemic, death is the result.

TREATMENT.

The patient must be quarantined; *quinine* administered daily (in gr. x. doses). *Counter irritation* should be applied over the region of the kidneys at the commencement of the attack, and *calomel* administered as a cathartic. It is better to combine the *calomel* with the *quinine*, giving gr. x. of each at a dose. Administer *milk and lime-water*, in order to overcome, if possible,

the nausea and vomiting. Should there be restlessness with tossing and rolling of the head, it is best controlled by the *hypodermic* use of *sulphate of morphine.* As soon as the stomach can receive food, the composition of the blood should be improved by giving a most nutritious diet combined with *wine, quinine,* and *iron.*

MALARIAL FEVERS.

In order to produce these fevers, there must be a certain amount of vegetable matter and moisture on the surface of or in the soil. A temperature ranging from 58° to 65° is required for its development. These fevers occur especially in marshy districts when covered by a thin sheet of water, and exposed directly to the sun, or when they have dried up; also when salt and fresh-water become mixed in the marshes; in damp-bottomed lands that are exposed to an annual overflow; or when new lands are opened up. The malarial poison may be conveyed by rivers which have their course in and flow through malarial districts, or it may be transmitted by the wind for a distance of about four miles and three-quarters. The limit for malarial fevers is 63° north and 57° south latitudes, seldom above an altitude of 2,500 feet. It can be prevented by free drainage, and may be arrested by cold. It is less virulent during the day than the night. The poison may be introduced into the system through the air we breathe and the food and water consumed. It is most liable to attack the weak, anæmic, and drunken. Taking cold over fatigue, sudden changes in the temperature, etc., are apt to predispose one to an attack.

SIMPLE INTERMITTENT FEVER.

DEFINITION.

Is a condition due to a malarial poison taken into the body through the lungs, food, etc.

MORBID ANATOMY.

The following are the constant pathological changes: Congestion of the internal organs; enlarged spleen and liver (due to hyperæmia); more or less hyperæmia of the kidneys and mucous membrane of the intestines.

ETIOLOGY.

The predisposing causes are intemperance; exposure to the night air; excessive fatigue; bad hygiene; and general depression. In children it is generally ushered in by convulsions.

TYPES.

(1) *Quotidian*—(most common) occurs in the morning. A paroxysm takes place every twenty-four hours, and lasts from eight to ten hours.

(2) *Tertian*—occurs about noon, with a paroxysm every forty-eight hours, and lasts from six to eight hours.

(3) *Quartan*—occurs in the afternoon or evening. A paroxysm takes place every seventy-two hours, and lasts from four to six hours.

(4) *Quotidian Double.*—There are two paroxysms daily; the one severe and the other mild.

(5) *Tertian Double.*—There are two paroxysms every second day; as to-day, a severe chill and slight fever; to-morrow, a severe fever and slight chill, and so on.

(6) Also a fever which occurs *every seventh day*, or *multiple of seven*.

STAGES.

Each of the types in this disease are characterized by a cold stage; a hot stage; and a sweating stage.

SYMPTOMS.

Cold stage—(lasts from half an hour to three hours).

Creeping coldness in the back, extending to the extremeties.
Headache—(supra-orbital).
Chills—lasting from half an hour to three or four hours.
Urine—abundant and pale.
Temperature—(elevated in the axilla or rectum).
Surface temperature is below normal.
Temperature per rectum is often 104°.
Livid appearance of the skin, face, etc.—eyes sunken.
The so-called "Goose-flesh" may exist, due to the erection of the papillæ of the derma.

Hot stage—(lasts from half an hour to two hours).

Temperature—rises suddenly 106°–107° in the axilla.

Pulse—rises.
Tongue—dry.
Great thirst.
Intense heat and redness of the skin.
Carotid pulsation.
Great restlessness and uneasiness.
Urine—high-colored and scanty; almost suppressed.

Sweating stage—(lasts from one to two hours).

Commences first on the forehead, when the patient goes off into a sleep, and wakes up much exhausted.
Temperature—is about normal.

DIFFERENTIAL DIAGNOSIS.

From remittent fever and pyæmia.

PROGNOSIS.

In the simple form the prognosis is good. If malarial cachexia be developed, there is the enlarged spleen, and liver, and pigmentation of tissue, which prejudices the life of the patient.

TREATMENT.

The patient must be kept in bed. *Opium* should be given in moderate doses *hypodermically* early in the cold stage, to diminish the severity of the attack. *Quinine*, gr. xxx., should be administered between the termination of one paroxysm and the hour when another is expected.

In the cold stage.—The patient should drink hot water, or something hot, and be covered with blankets, and hot water bottles applied to various parts of the body.

In the hot stage.—The blankets should gradually be removed, and the patient be given cold drinks. When there is vomiting, *opium* should be used *hypodermically*.

In the sweating stage.—The patient must be left alone. In order to overcome the paroxysms, give *quinine*, gr. xxx., one our previous to the expected paroxysm, if possible, in the form of a solution, as follows: gr. x. just before the sweating stage (or, gr iij. hypodermically), and gr. xx. one hour before the paroxysm.

Keep the patient under cinchonism for a few days, or even one month, by giving *quinine*, gr. xv. every day, about two hours before the expected paroxysm. Remove the patient if possible, from the district.

It is always of great advantage to give a good dose of *calomel*, gr. x., and *jalap*, gr. xv. at the commencement of the illness, as it will oftentimes avert the paroxysms; then resort to the *quinine* after the bowels have been evacuated. *Pilocarpine*, gr. $\frac{1}{6}$, may be given *hypodermically*, to break up an attack. If the *quinine* does not act, commence by giving *Fowler's solution*, gtt. v., and increase the dose one drop every day till you reach gtt. xxx.; having attained this maximum, commence again with gtt. xv. *Piloridzine*, gr. xv.–xx. (the active principle of the apple-tree bark) may be given before each meal in cases *where quinine has failed* to afford relief. This drug is very little known, but has proved successful in the hands of many.

PERNICIOUS FEVER.

SYNONYMS.

Congestive; climatic; ardent; African; tropical typhoid.

DEFINITION.

It is the most severe and dangerous form of malarial fever. It may assume any of the types of a periodical fever, but the quotidian and tertian are the most common forms.

VARIETIES.

Comatose; delirious; gastro-enteric; algid; icteric; hemorrhagic; and colliquative.

MORBID ANATOMY.

Is similar to the changes of intermittent and remittent fevers, but differs very much in degree, and will depend upon the severity of the fever. In the blood, the pigmentation is more abundant, and the pigment material may be in the form of granules, or plates, or have a cellular outline. On *post-mortem*, in some in-

stances, the brain-substance will be more or less stained with pigment material; in others, minute blood extravasations, scattered throughout the substance of the organs, will be detected. Small blood extravasations into the spinal cord, with more or less pigmentation, may produce tetanic spasms during life. Every organ in the body may be pigmented. The various organs may be markedly engorged. The spleen may be found softened, as well as enlarged, or it may have infarctions scattered through its substance. The kidney and lungs are sometimes the seat of extensive hyperæmia, causing great interference in their functions.

ETIOLOGY.

It differs from the simple forms of malaria only in degree. It prevails only in localities where the average range of temperature is 65°.

SYMPTOMS.

These frequently commence in the same manner as in intermittent or remittent fever.

Comatose variety.

After a remission, the patient passes into a state of stupor.

Face—flushed.

Conjunctiva—congested.

Pupils—dilated.

Respiration—slow, deep, stertorous, and, later on, increased in frequency.

Pulse—slow (which becomes frequent as the disease advances).

Temperature—105° to 107° (in the axilla).

Urine—retained.

Bowels—move involuntarily.

It is usual, in about twelve hours, for a remission to take place, and the patient to awake, in a profuse perspiration, to consciousness; but, with the next exacerbation, the same symptoms appear only with increased severity, and the patient passes into a fatal coma.

Delirious variety.

Delirium—violent in character.

Face—flushed.

Carotid pulsation.
Eyes—injected.
Tetanic spasms—may occur.
Conjunctiva—congested.
Memory of what has transpired seems lost.
Pupils—dilated.
Distinct intermission—may occur.
Pulse—full and hard.
Temperature—107° to 108°.

Attacks of delirium may be repeated three or four times.

Gastro-enteric variety.

Vomiting—excessive and purging. The materials vomited and discharged from the bowels are blood-stained.
Sense of weight and burning in the stomach.
Cramps—in the calves of the legs.
Coldness and blueness of the surface.
Pulse—small and almost imperceptible.
Eyes—sunken.
Face—resembling that of cholera.
Thirst—intense.
Inspirations—double, followed by a double sighing expiration.

When fully developed, few patients recover.

Algid variety.

Surface of the body is cold.
Rectal temperature—104° to 107°.
Sensation of burning.
Thirst—intense.
Cold perspiration covers the body.
Pulse—slow, and finally disappears at the wrist.
Tongue—white, moist, and cold.
Breath—cold. Voice—feeble and sunken.

When once this type is established, it progresses to a fatal termination, unless arrested early by treatment.

Icteric variety.—This stage usually lasts three hours, and often terminates in death.

Chill—long and continuous.
Jaundice—appears first in the eye, but rapidly passes over the entire body.

Copious vomiting of bile and a bilious diarrhœa.
Headache—intense.
Delirium—low and muttering in type.
Pain—in the region of the liver and spleen, and over the kidneys.
Feeling of numbness in the limbs.
Pulse—small, frequent, and hard.
Urine—deep-colored.

As the hot stage approaches, the
Pulse—becomes more frequent.
Respiration—labored.
Temperature—106° to 107°.
Thirst—intense.

If the sweating stage is reached, recovery usually takes place.
Death usually occurs during two or three paroxysms.

DIFFERENTIAL DIAGNOSIS.

The comatose and delirious varieties may be confounded with cerebral apoplexy, meningitis, or acute uræmia; the gastro-enteric and algid with Asiatic cholera; and the icteric with yellow fever.

Cerebral apoplexy.
Hemiplegia—is usually present.
Coma—sudden.
Pulse—slow.

Comatose and delirious varieties.
Hemiplegia—rare; but, if present, occurs late.
Coma—later; and, with the coma, a rise in temperature.
Pulse—rapid.

Asiatic cholera.
Vomiting—projectile.
Discharges—rice-watery.
Urine—albuminous.
Temperature—not so high as in the gastro-enteric and algid varieties.

Gastro-enteric and algid varieties.
Vomiting—excessive and blood-stained.

Discharges—blood-stained.
Urine—does not contain albumen.

Yellow fever.

New comers are almost certain to contract the disease.
Jaundice—late.
Urine—rarely bloody.

Icteric variety.

Those are seized who have been longest under the influence of the malarial poison.
Jaundice—early.
Urine—frequently contains blood.

PROGNOSIS.

Is unfavorable, unless you can control the disease before the second paroxysm comes on.

TREATMENT.

On account of the alarming symptoms which follow so rapidly on each other, prompt and vigorous measures must be resorted to. Depletion in every form should never be practised. The only remedies which can be relied upon for controlling every variety of this disease are *quinine* and *opium*, which should both be given *hypodermically*. The following formulæ for the solution of *quinine* are those employed by the English surgeons for this disease: ℞ quiniæ disulph., gr. l.; acid. sulph., ♏ v.; acid. carbolici, ♏ ij.; aquæ dest., ʒ i.; or, ℞ quiniæ sulph., ʒ i.; acid. hydrobromici, ʒ ij.; aquæ dest., ʒ vi. Thirty minims represent from three to four grains of *quinine*. From five to seven grains should be given every hour till the paroxysm has passed; after that, it should be given in three grain-doses every four hours. One-fourth of a grain of *morphine* should be administered with the first injection of *quinine*, and continued until the patient is brought fully under its influence.

Warburg's tincture is claimed by many to have more control over this form of fever than *quinine* or *morphia*. One-half ounce of this mixture is given undiluted after the bowels have been evacuated by a convenient purgative, drinks of all kinds being withheld, and in three or four hours after, the other half is ad-

ministered in the same way, when profuse, but not exhaustive perspiration, rapid decline of temperature, and abatement of headache take place. A second ounce of this tincture is seldom, if ever, required.

DENGUE FEVER.

SYNONYMS.

"Break-bone fever;" and "dandy" fever.

MORBID ANATOMY.

Is the same as that of the severer types of malaria, excepting that there is an exanthematous eruption which commences on the palms of the hands, extending rapidly over the entire body; it is also accompanied by arthritic changes, and enlargement of the external lymphatic glands.

ETIOLOGY.

It may be either epidemic or sporadic, and attacks all classes and ages. It is a portable disease (some say it is contagious, which is questionable). It may be the precursor of yellow fever. It occurs particularly in the Southern States of America, and the severity of the attack depends upon the intensity of the poison. Its period of incubation is from three to five days.

STAGES.

Initiatory; fever; remission; and secondary fever and convalescence.

SYMPTOMS.

Initiatory Stage.

Headache.
Photophobia.
Great restlessness.
Chilliness.
Pain—in the back, limbs, and joints.
Swelling of the small joints, and soreness of muscles.
Fever—which lasts from twelve hours to four days.

Temperature—may reach 107°.
Pulse—120 to 140.
Face—flushed.
Eyes—red and watery.

Stage of Fever.

Pain increases after the fever has lasted twelve hours and extends down the sciatic nerve.
Nausea and vomiting.
Pain—in the epigastrium.
Glands—swollen (axillary, inguinal, and cervical).
Epididymitis—in some cases.

Stage of Remission.

Profuse sweatings—may occur.
Diarrhœa—may occur.
Epistaxis—may occur.
Pains, etc.—diminish.

Stage of Secondary Fever and Convalescence.

Fever, and pains, etc.—return with greater intensity.
Eruption—appears about the fifth day, first on the palms of the hands, then on the neck, and spreads. It is of a papillary form, resembling scarlet fever.
Tongue—is coated and dark-brown (like in typhoid fever).
Heart—becomes intermittent.
Glands—may suppurate.

DIFFERENTIAL DIAGNOSIS.

From rheumatism, remittent and relapsing fevers.

From rheumatism—by the febrile symptoms which precede the arthritic pains, and the fact that the fever prevails epidemically.

From remittent fever—by the persistency of the rheumatic and neuralgic pains, eruption, and character of the remissions.

PROGNOSIS.

Is usually favorable. Is only unfavorable in the aged, and feeble infant.

TREATMENT.

It is customary in this disease to administer *ipecac*, *calomel*, and *colchicum* every night in cathartic doses. *Calomel* should never

be given alone if its specific effects are likely to be produced. In order to relieve the pain in the head and limbs, *colchicum* should be given in combination with *spirits of nitre* and *nitrate of potash* (as a diaphoretic), in connection with an effervescing draught. During the remission, keep the bowels open by a saline cathartic, and give *quinine* combined with an alkali at stated intervals. For insomnia, *narcotics* may be given in small doses. Should there be exhaustion, the free use of wine and beer may be resorted to, and nutritious diet administered at stated intervals, day and night. The lymphatic enlargements (if any) should be painted with *iodine*.

Citrate of quinine and iron will be found of great service during convalescence. Should the joints remain swollen and tender for a long time, blistering the parts has been recommended.

TYPHO-MALARIAL FEVER.

DEFINITION.

Is a fever produced by the combined action of a septic and malarial poison.

MORBID ANATOMY.

The changes which occur in this disease are confined to the blood, and the various internal organs.

Blood.—The changes that occur in the vital fluid are similar to those in typhoid fever, combined with the presence of free pigment granules.

Liver.—Is increased in size, and, *on section*, resembles the nutmeg-liver. It may resemble the liver of yellow fever or the bronzed liver of remittent fever. The *microscope* will show free fat, and brown pigment granules in the hepatic cells.

Spleen.—Is enlarged, softened, and almost black in color. The Malpighian bodies are prominent.

Kidneys.—There will be found a state of marked hyperæmia in the cortical substance.

Lungs.—There will be a hypostatic congestion at the most dependent portion of each organ.

Heart.—Will be pale and flabby. There will be a granular degeneration of its muscular fibres. In its cavities will be found exsanguinated clots.

Intestinal lesions.—As the follicles enlarge, there is a tendency to the deposit of a black pigment. Extensive ulcerations of these follicles also take place, and they may invade the adjacent mucous membrane. These ulcerations are found principally in the ileum, and involving Peyer's patches; their edges are irregular and everted, and their base is of a grayish color, often mottled with dark spots. They may extend into the sub-mucous tissue, involve the muscular coat of the intestine, and even perforate the peritoneal covering.

ETIOLOGY.

It is only met with in malarial districts. Its development is favored by bad hygienic conditions, such as improper diet, overcrowding, bad sewerage, etc.; it is never propagated by contact, or by morbid excretions, but it is a distinct malarial poison. It is really a combination of two well-recognized forms of fever.

SYMPTOMS.

When the malarial element is predominant.

Distinct chill, malaise, and pain in the limbs and back.
Countenance—waxy, clay-colored, or of a yellowish tinge.
Temperature—rises to 103° to 104° in a few hours.
Pulse—full and forcible (usually about 100).
Mental disturbance.
Delirium—sometimes.
Tenderness in the right iliac fossa.
Nausea.
Vomiting.
Epigastric tenderness.
Diarrhœa—usually present, and may precede the chill.
Tongue—is at first pale, flabby, and has a smooth surface; it soon becomes covered with a yellowish-white coating; later, it becomes red and with a brownish coating, and in severe cases, it may suddenly become clear, red, and shining. Sordes may collect on the teeth and lips.

In fatal cases—towards the second or third week, the symptoms resemble those of fatal typhoid fever, and the pulse reaches 130-140; is feeble and irregular; and coma and death ensue. In cases that recover, signs of amendment may be noticed about the tenth or twelfth day.

When the septic element is predominant.

General malaise.

Chill — is distinct, or a complete intermittent or remittent paroxysm may be produced.

Temperature—may rise gradually or suddenly to 104° to 105° within twenty-four hours.

Remissions occur on every second or third day.

Hepatic tenderness.

Spleen—enlarged.

Pulse—first week full, rarely above 100; but. during the second and third week, is small and compressible; and, in severe cases, it becomes intermittent (ranging from 110 to 130).

Tongue—swollen at first, with red projecting papillæ and a light white coating; afterward it resembles closely the tongue in typhoid fever.

The surface of the body becomes dry and harsh.

Skin—is of a bronzed hue.

Jaundice—well marked.

Urine—high-colored and scanty.

Diarrhœa—may occur at any period, but is not usually excessive until the second or third week. The discharges have a very fœtid odor, are watery and dark-colored, and, in the later stages, may contain blood.

Delirium—(which follows the headache) may be present, and is muttering in character, and other typhoid symptoms.

DIFFERENTIAL DIAGNOSIS.

It may be confounded with typhoid, remittent, relapsing, typhus, and yellow fevers.

Typho-malarial fever.

The advent is marked by a distinct chill; the rise in tempera-

ture is sudden; there is a peculiar eruption which remains throughout the course of the fevers; there is a distinct periodicity in the febrile action; a jaundiced hue to the surface; and the blood contains free pigment.

Typhoid fever.

The symptoms come on insidiously; there is a typical range of temperature; on the sixth or eighth day a *rose-colored eruption* appears.

Typho-malarial fever.

There will be enteric symptoms early; typhoid symptoms are developed as the fever advances.

Remittent fever.

There are no enteric symptoms, but it yields more promptly to the use of quinine than typho-malarial fever.

Typho-malarial fever may be easily diagnosed *from yellow fever*, as, in the latter, the range of temperature is lower, and falls suddenly on the third or fourth day; there will be also the circumorbital pain; the appearance of the eye; the peculiar color of the skin; the character of the matter vomited; the absence of diarrhœa; the presence of albumen in the urine; and, finally, by the fact, that it is a portable disease, and prevails epidemically.

PROGNOSIS.

The septic is more fatal than the malarial type. The hygienic surroundings, previous habits of the patient, and range of atmospheric temperature will of course aid your prognosis. The average mortality is given as one in every twelve or twenty-four. The average duration of cases terminating in recovery is from three to four weeks. Capillary bronchitis and pneumonia are very dangerous when developed during the third week.

TREATMENT.

Depends entirely upon the type of the disease. Prevent the overcrowding in the houses; remove your patient, if possible, from his present surroundings; improve his diet thoroughly; and by these means you will prevent the development of a poison which gives to this fever its typhoid type. In cases where the malarial ele-

ment is prevalent, administer *quinine* (grs. xx.–xxx.) in two or three doses of gr. x. each every hour until the desired quantity has been given. In the septic variety, quinine has no effect. By some, *arsenic* is claimed to have a specific influence over this form of the disease, but its beneficial effect is due to its power over malarial affection, and not to its specific influence over the fever. In fact, the treatment may be said to be almost identical with that of typhoid fever.

TYPHUS FEVER.

SYNONYMS.

Ship fever; hospital fever; jail fever; camp fever; petechial fever; putrid fever; continued fever; fourteen days' fever; cerebral typhus; exanthematous fever.

MORBID ANATOMY.

The first changes that take place are in the blood, which is darker than normal, and when drawn during life, coagulates imperfectly or not at all; but, if it does, the clot is of the consistency of putty. The fibrin is diminished and loses its power of coagulation. The red globules first increase, and then diminish, and urea and ammonia are found to be in excess. The blood when drawn rapidly undergoes ammoniacal decomposition.

The *microscope* shows the red blood-globules to have lost their outline; they have serrated and irregular edges, and undergo degeneration. The coloring matter passes through the walls of the vessels and stains surrounding tissues.

Parenchymatous degeneration next takes place. The blood rapidly undergoes decomposition after death, even sooner than in typhoid fever, often beginning before death. The muscles are of a brownish color, dry, and granular. The liver and spleen undergo similar degeneration. In the kidneys, the changes are much more extensive and constant than in typhoid fever; the cortical substance is swollen, opaque, and fatty, and is marked by a cloudy swelling of the epithelium in the renal tubes. This cloudy swelling also occurs in the heart-fibres, causing flaccidity, and giving

to the heart a dark-brown color. There is often serum in the pericardium; clots occur in the cavities of the heart, and thrombi on the walls of the veins. Ulceration of the mucous membranes of the mouth and larynx ensues, involving the sub-mucous tissues. Splenization of the lungs takes place, and the cerebral vessels become congested. The sinuses and blood-vessels in the brain are engorged, so that they will stand out prominently when the calvaria is removed, and there will be more or less effusion in the meshes of the pia mater (often from eight to ten ounces). If the effusion be turbid, it is an indication that there has been meningitis. If the effusion be abundant, the sulci are deepened and the convolutions are sharpened in outline.

Abdominal lesions.—Intestinal changes are absent. There is no ulceration of the glands excepting changes which have taken place from congestion (causing the shaven-beard appearance in Peyer's patches).

Complications.—These may be pulmonary, cerebral, or spinal.

The *pulmonary* include bronchitis, catarrhal pneumonia, pleurisy, congestion, œdema, pulmonary gangrene (from pneumonia), and laryngitis (from extensive bronchitis).

The *cerebral* or *spinal* include meningeal inflammation with serum in the meshes of the pia mater; but, besides this sub-arachnoidean effusion, there must be also plastic exudation; the arachnoid has lost its shining color and is thicker than normal.

There will be active delirium; contracted pupils; pulse, slow, full, and then rapid, and irregular, and intermittent, and the urine is often passed with difficulty. The parotid, sub-lingual, and glands of the neck become greatly enlarged, so as to interfere with deglutition and to often cause death. The inguinal glands may become so enlarged as to interfere with the return circulation. There may be a swelling of the lower limbs from degeneration and feebleness of the heart, causing retarding of the circulation and thrombi in the superficial veins. Suppuration and cellulitis may occur, resulting in extensive abscesses.

SYNOPSIS OF THE FOREGOING PATHOLOGICAL CHANGES.

The changes that take place occur in the skin, organs, and blood.

In the skin—the *mulberry rash* will be present on the side of the chest and abdomen, and possibly on the extremities; it disappears on pressure (for three days) and the spots are dark in color and somewhat elevated.

In the brain—there is hyperæmia of the vessels of the convexity; meningeal exudations: serous effusion into the meshes of the pia mater and ventricles; the arachnoid becomes opaque and dotted with yellowish spots.

In the alimentary canal—changes take place in Peyer's patches (producing the "shaven-beard" appearance).

Complications.—Bronchitis, pneumonia, splenization of the lung, pleurisy, œdema, meningitis, parenchymatous nephritis, and finally ulceration of the larynx, pharynx, and mouth, and enlarged cervical and probably inguinal glands.

In the blood.—All the common lesions of fevers (see page 316), and a marked increase in urea.

ETIOLOGY.

This disease is due to a specific poison, *communicated* from the sick to the healthy *by contagion* only. Over-crowding and bad ventilation favor the spread and increase the severity of the disease. The poison passes into the blood mainly through respired air.

SYMPTOMS.

Initial symptoms—come on suddenly, and include:

Vertigo.
Loss of appetite.
Chill.
Steadily increasing frontal headache.
Pain—in all the limbs (especially the thighs).
Extreme prostration.
Tottering gait—the patient is then compelled to take to his bed.

1*st Week.*—Temperature—104°-105°, morning and evening variations are slight; it rises one or two degrees.

2*d Week.*—Temperature—is liable to sudden depression, but generally falls at this period. If it continue, it denotes development of cerebral symptoms, commencing with this sudden depression. It is usually highest on the second or third day of the disease.

Tongue—is first swollen, white, and coated; then of a yellowish-brown color, and thick; and, later on, becomes dark, dry and fissured.

Delirium—low, muttering, or active (often becomes furious about the eighth day); it comes on early and continues till the end of the disease.

Coma-vigil—patient has horrid fancies and visions, and is absolutely indifferent as to what is going on, and lies with his eyes wide open. Is usually followed by a fatal termination.

Headache—severe and persistent for the first ten days.

Stupor and somnolence—seldom absent.

Paralysis of the sphincters of the anus and bladder or, of the mucous coat of the bladder (causing retention).

Dysphagia and partial or complete aphonia.

Picking at the bed clothes, and subsultus tendinum.

Heart—the first sound may be inaudible.

Pulse—during the second week the pulse may be small and feeble 140 or 150, which is unfavorable; is generally 100, frequent, soft, rapid, easily compressible, and irregular: if 120 for three days in the first week, it denotes danger.

Urine—is first diminished, and then increases, and will contain a small amount of albumen; or, if it be a severe case, albumen will be large in quantity, and there will be renal casts, also epithelium and fatty casts of the uriniferous tubes.

Eruption—(*called the mulberry rash*)—consists of small, irregular-shaped, dark, brick-dust spots; is slightly elevated at first, and remains throughout the disease. It appears on the fifth to the seventh day of the fever, and disappears on pressure (for the two first days of the eruption). Is present upon the sides of the chest, abdomen, and possibly on the extremeties.

Constipation.
Countenance—dull, heavy, and, later on, of a *mahogany* color.

CAUSES OF DEATH.

Death may result from meningitis, pneumonia, capillary bronchitis, gangrene, exhaustion from the effects of poison, Bright's granular kidney, coma and syncope.

DIFFERENTIAL DIAGNOSIS.

From typhoid and relapsing fevers; measles; pneumonia; acute Bright's disease; meningitis; delirium tremens; erysipelas; pyæmia; and septicæmia.

PROGNOSIS.

Is always grave. It depends upon the age of the patient, the character of the epidemic, and the complications. With the intemperate, it is likely to prove fatal.

In cases where debility exists from advanced age, intemperate habits, privation, previous disease, mental depression, over-crowding, bad ventilation, and gouty diathesis, the prognosis is always dangerous.

TREATMENT.

Quarantine your patient, as it is a contagious disease. Thoroughly disinfect the premises for two or three days, at the time and after the disease. The patients should be placed in pavilions or tents in order that they may have *plenty of fresh air*. Remove all windows regardless of cold, and supply plenty of blankets.

Medical treatment.—Fresh air is the only remedial agent. First reduce the temperature, and then sustain the heart's power. Administer *quinine* and apply cold to the surface. Milk should be the only diet, and water may be drank freely. If the temperature be 104°, put the patient in a bath 10° lower than the temperature of the body. Ice or cold water may be added to the bath till the temperature falls to 68° or 70°, and the patient kept in it till his temperature falls to 101° or 102°; then he should be taken out, dried quickely, and put into bed. Should the temperature rise again, repeat the bath. If there should be intense pain and delirium after taking the bath, put ice-bags to the head.

Give *quinine*—gr. lx. in two hours, with gr. x. *pulverized digitalis* added.

Stimulants (?).—Patients under twenty-five years of age rarely require it unless they happen to have been intemperate prior to the attack. To the old and feeble, when occasionally administered, it probably does good. You should always watch your patient when administering stimulants, as in typhoid fever. One ounce in twenty-four hours is all that is necessary, and then only to sustain the heart's power.

Cardiac sedatives.—Veratrum, aconite, and *digitalis* may be administered. For failure of the heart's power, give *digitalis* ʒ iv.–vi. in twenty-four hours, or *quinine* and *digitalis* in the same proportions.

For insomnia—keep the room dark and quiet, give opiates in full doses, and apply cold to the head.

For insomnia and delirium—give *chloral* gr. x.–xv.

For stupor—if occurring early, give coffee, musk, camphor, or cold douche.

For coma—*valerian* and *phosphorus* may be given, but they are not efficacious.

If the patient cannot take *quinine* by the mouth, give it by the *rectum,* about one-third more than by the mouth, thus, quinine dissolved in acid causes it to become disulphide or in the form of a suppository of the bisulphide by the rectum.

REMITTENT FEVER.

SYNONYMS.

Southern; Western; African; continued; bilious; and acclimative.

DEFINITION.

Is one of the malarial fevers, characterized by a sudden invasion and persistent high febrile symptoms; temperature frequently rising to 105°–106°.

MORBID ANATOMY.

It is the result of malarial poisoning; consequently there is diminution in the red blood-corpuscles; and pigment granules exist (due to hœmoglobin which has escaped from the red blood-corpuscles in the vessels, and developed in the liguor sanguinis,

and which may infiltrate into the adjacent tissues and cells). The changes resemble those occurring in intermittent fever. The principal lesions will be found in the spleen, liver, stomach and intestines.

Spleen.—Is enlarged and bronzed (though not so much as in intermittent fever), from congestion of the vessels, and an excess of pigment.

Liver.—(called the "bronzed liver") is not much increased in size. The peculiar color is due to pigmentation of the tissues throughout the organ. This is *the characteristic lesion* of the fever.

Stomach.—The mucous membrane is more or less congested, thickened, and softened.

Intestines.—The mucous membrane is more or less congested thickened, and softened. Peyer's patches are enlarged and have the "shaven beard" appearance. Sometimes ulceration of the intestines occurs, or there may be simple hyperæmia of the mesenteric glands, due to a catarrhal inflammation.

ETIOLOGY.

The predisposing cause is malarial fever. Remittent fever may pass into intermittent during convalescence. It occurs chiefly in 63° north and 57° south latitude. It prevails along the banks of rivers. The miasm may be conveyed by the winds. It occurs in marshy regions where there is but little water.

SYMPTOMS.

Remittent Fever.

Premonitory symptoms.

Oppression in the epigastrium.

General malaise and *sudden* development of symptoms.

Cold stage.

Chill—short and not severe (a general coldness).

Temperature—rises about 3° above normal during the chill.

Headache—intense.

Pain in the back and limbs.

Hot stage.

Fever—which follows the chill, when temperature rises rapidly and within 12 hours may have reached 105° or 106°.

Pulse—increases with the temperature (reaches 110 to 130).
Face—flushed.
Eyes—suffused.
Conjunctiva—conjested.
Great restlessness.
Nausea—persistent and distressing.
Vomiting—consists first of contents of the stomach, then stringy mucus tinged with greenish matter, and finally a slight black vomit.
Great oppression and tenderness in the epigastrium, unrelieved by vomiting.
Symptoms increase in severity for 12 *hours, when follows the Remission stage.*
Perspiration—not profuse—commences on the forehead, extending over the entire body.
Temperature—falls 1° or 2° (but there are still evidences of fever, as it does not reach normal).
Pulse—suddenly falls 10 or 20 degrees.
Thirst—diminishes.
Vomiting—decreases.
Period of exacerbation—occurs at the same time on the following day when all symptoms return with increased severity. This constant change may continue for a week and the fever becomes a continued one when
Tongue—may be parched and sordes may be present upon the teeth.
Countenance—dull and heavy.
Distress and *pain* and *tenderness in the epigastrium.*
Constipation—which may give way to diarrhœal discharges of a brownish character with fullness of the stomach and local tympanites.
Typhoid symptoms—then come on as in the third week of typhoid fever.

Bilious Remittent.

Vomiting—bilious.
Jaundice of the skin—like yellow fever.
Tenderness over the region of the liver.

DIFFERENTIAL DIAGNOSIS.

From intermittent fever ; typhoid fever ; bilious remittent fever; yellow fever ; pyæmia; septicæmia and typho-malarial fever.

PROGNOSIS.

In a simple case the prognosis is always good. Even cases of severe types should recover if properly managed. The average duration of this fever is two weeks.

TREATMENT.

The patient must be placed under the best possible hygienic surroundings, and the same arrangements carried out respecting the sick room as in typhoid fever. *Quinine* must be given during the exacerbrtion (paroxysm) or remission, in doses varying from *gr. x-xx* according to the severity of the fever, and repeated every two hours till cinchonism is produced. If, after the free use of quinine the exacerbations be more severe, the remissions less frequent and typhoid symptoms manifesting themselves, *stimulants* may be resorted to, and probably in large doses, while passing through this period.

The surface of the body should be sponged with cold water if the exacerbations be intense, the headache very severe and the restlessness or fever are not relieved by the quinine given in large doses.

SMALL-POX.

SYNONYM.

"Variola" or "varioloid."

VARIETIES.

Variola discreta (separated variola).

Variola confluens (the spots running into each other).

Variola hemorrhagica (the eruption being black, on account of hemorrhagic effusions).

MORBID ANATOMY.

1st. There is congestion of the organs, as of the brain, lungs, liver, spleen, and kidneys.

2d. There is the state of granular or acute fatty degeneration (resembling changes due to phosphorus poisoning).

3d. There is the state of fatty degeneration of the liver, kidneys, and heart (which is usually yellow, flabby, and brittle).

4th. There are small hemorrhages into nearly every viscera, with ecchymosis of the serous membranes and fluid blood in all the cavities.

The *characteristic lesion* is the eruption upon the mucous membrane and skin. At first, a *congestion of the papilla*, sometimes uniform, and sometimes in spots, giving rise to little red spots on the surface, may be perceived, which are surrounded with cells rapidly undergoing granular degeneration. These papules are due to changes in the surrounding cells in the rete Malpighii, and in the capillaries, and to certain new cell infiltrations.

A serous infiltration on the surface of the papule now develops, called *the vesicle* (consisting of blood serum, which has escaped through the walls of the capillaries). The centre of the vesicle *becomes umbilicated* (depressed), due, probably, to the serous infiltration taking place more rapidly at the periphery of the vesicle than at the centre, causing elevation of the periphery, although it has been explained in other ways (adhesion of centre of vesicle to the hair of the skin, formation of lymph-bands in the vesicle, etc.).

The vesicle next *changes color*, due to migration of white blood and pus corpuscles from the capillaries into the surrounding tissue, and then *becomes a pustule*. Destruction of the tissues at the point where the papillary congestion first occurred takes place. If the superficial layer of the skin be only affected, there is no scar left; but, if the inflammation extend to the deeper cellular tissue, *sloughing* occurs, *resulting in cicatrix and pitting*.

After the pustules have formed, the inflammatory products dry down, and a crust is formed, which contracts in the central portion. These crusts separate when the inflammatory process subsides, and recovery takes place. These lesions are not confined to

the skin, as they may also form on the mucous membrane of the stomach, intestines, bronchial tubes, larynx, and conjunctiva, urethra, etc.

SYNOPSIS OF THE FOREGOING.

The lesions of this disease occur in the skin, mucous membrane, organs, and blood.

In the skin.—All the stages of the eruption may exist, which include the macule, papule, vesicle, umbilicated vesicle, pustule, scab, or cicatrix; also, possible abscess, ulceration, and local sloughs.

In the mucous membrane.—The same conditions are possible in the urethra, eye, nose, mouth, alimentary canal, and middle ear.

In the organs. — Parenchymatous nephritis, œdema glottidis, evidences of complications, as pleurisy, pneumonia, peri- and endo-carditis, meningitis, and bronchitis, and fluid blood, may be found in some of the capsules.

In the blood.—The common lesions of fevers (see page 316).

ETIOLOGY.

It is propagated only by contagion; by means of the breath and exhalations from the skin; the poison may pass through the atmosphere for a distance of about two and a half feet in the open air. It may be contracted by entering a contaminated room. It can be conveyed by clothing, but the clothing must contain pus or crusts from the small-pox virus. It attacks all periods of life.

Stage of infection.—This may occur during the period of suppuration, or the stage of desiccation, or even during incubation. The poison is absorbed by the mucous membrane of the respiratory tract during respiration.

The *period of incubation* varies from ten to thirteen days; if the poison be introduced from inoculation, forty-eight hours may only elapse before the stage of initiatory fever takes place.

STAGES.

Incubation. Invasion. Eruption. Suppuration. Desiccation.

SYMPTOMS.

VARIOLA DISCRETA.

Period of incubation and invasion.—(The invasion may be sudden or gradual.)

Chill and pain in the head, back and loins.
Temperature—elevated, 104°–105° (may reach as high as 107°).
Pulse—may rise from 100 to 120, and even 140 per minute, and may be full or more frequent.
Nausea and vomiting.
Soreness of the throat and pain in the pharynx.
Delirium—about the third day (may occur).
Face—flushed.
Photophobia.
Headache.
Throbbing of the carotids.
Conjunctiva—congested.
Great restlessness.
Countenance—anxious and flushed.
Somnolence.
Respiration—short, frequent, and labored.
Cough—stridulous.
Voice—husky, if the larynx be involved.
Vertigo and convulsions (in children).
Excessive languor.
Anorexia.

Stage of eruption—on the third day after the initial fever.

Eruption—usually commences first along the edges of the hair; next on the forehead, nose, and upper lip; then on the body and extremities, a day or two later; and, lastly, on the hands and feet. It is of a *pale-red color*, resembling *flea bites*, and about the size of a millet-seed or pin's head; there is a sense of burning or itching on the surface, and it is less abundant on the body and extremities than on the face. On the second day, it becomes darker, elevated, and papular. On the third day, it becomes more conical, and a vesicle forms at its apex. On the fourth and fifth days, it is spherical and about the size

of a pea, and as it enlarges, becomes umbilicated, the fever then subsides, and all pain is gone.

Stage of suppuration, or secondary fever,

Suppuration—begins about the eighth day of the eruption with the following symptoms :

Chill.

Temperature—rises rapidly, perhaps higher than it did during the initial fever, sometimes as high as 108°-109°; it reaches its maximum when suppuration is at its height (about the ninth day) on the extremeties.

Pulse—becomes frequent.

Face—is a shapeless mass and swollen (if the eruption be abundant).

Itching—*intolerable.*

Breath—sweetish and of a sickly odor.

Skin—becomes red and tumefied; and if the spots be thickly set, they become confluent.

Stage of desiccation.

Desiccation—commences from the eleventh to the fourteenth day of the eruption, when the pustule either ruptures, discharges its contents, dries up and forms a yellow crust, or shrivels and dries up without rupturing.

The symptoms abate.

Scabs—come off (about the eleventh to the fourteenth day).

Skin—is of a red-brown color (lasting from five to six weeks).

Appetite—returns.

Convalescence—is established.

VARIOLA CONFLUENS.

The duration is short and severe, only lasting about forty-eight hours.

Stage of eruption.

Eruption—appears simultaneously all over the body; the spots are very numerous, especially on the face and hands. On the first day they are almost confluent; on the second day the skin is intensely red, and swollen, and vesicles form which become confluent. It may involve the mucous membrane of the mouth, throat, nares, and larynx, resulting in impossible deglutition.

Temperature—rises to 103°–104° till suppuration takes place.
Typhoid symptoms set in.
Tongue—becomes dry.
Patient is semi-comatosed with sub sultus, low delirium, and intense nervous depression.
Urine—albuminous.
Glands—parotid and sublingual enlargements may occur.
Œdema glottidis—may suddenly develop, which will cause death, unless laryngotomy be performed early.

Stage of suppuration.

Suppuration—steadily follows, forming flattened yellow-colored confluent patches, which run together. Large bullæ filled with sero-purulent fluid are thus produced, and these may spread over the face so as to render the patient perfectly unrecognizable.
Temperature—is higher than in the stage of eruption.
Vomiting—may be violent.
Diarrhœa.
Delirium.
Coma.

Stage of desiccation.

Desiccation—is slowly reached; large concentric crusts are formed, adhering to the skin, and beneath there is suppuration of the papillary layer, with extensive destruction of the true skin, causing ugly pits and scars.
The initial fever often reaches 106° to 107°, and in severe types 110°, and pulse is frequent and feeble.

COMPLICATIONS.

Inflammation of the serous membranes, especially pleurisy, peri-carditis, croupous and catarrhal pneumonia, severe bronchial inflammations, and uræmia.

Variola hemorrhagica.

This is a modified form of the other two varieties, only differing in the

Eruption—which is very dark over the whole body. There is also

Hemorrhage—from the various mucous membranes of the body; and in females profuse menorrhagia.

Suppuration—is seldom reached.

Death—results from exhaustion, due to hemorrhage, or from the effects of the small-pox poisoning.

CAUSES OF DEATH.

Death from small-pox may occur from œdema glottidis; general bronchitis; pneumonia; or acute fatty degeneration of the kidneys.

DIFFERENTIAL DIAGNOSIS.

It is more prudent to wait till the third day of the fever before venturing on a diagnosis, when the eruption will be well out and the vesicle found. It may be mistaken for measles, typhus, and meningitis, petechial eruptions, and chicken pox.

Small-pox—commences with chill and elevation of temperature; the temperature is very high, 106°–107°, and falls as soon as the eruption appears.

Measles—commences with coryza, sneezing, redness, and suffusion of the eyes; the temperature rises after the eruption appears.

Small-pox—the eruption appears on the third day, and is first seen on the face and forehead.

Typhus fever—(is difficult) there is greater loss of muscular power, and the eruption is first seen on the abdomen.

In Small-pox—the face is pale, and bears an anxious look.

In Meningitis—the face is flushed and deepens.

PROGNOSIS.

Depends upon the eruption, type of the disease, and age of the patient. *In variola discreta*—unless complications exist, recovery usually takes place (from one to five die). *In variola confluens*—the prognosis is more grave; one-half of the cases are generally fatal. *In variola hemorrhagica*—is usually fatal in forty-eight hours; and is more fatal in summer than in winter. If the patient has been of intemperate habits, is syphilitic, or a chronic case, the prognosis is bad. Whenever there is an abundance of the eruption, the prognosis is bad. When the urine becomes scanty

and high-colored at the commencement of the stage of suppuration, you may be sure there are kidney complications, and convulsions may occur, resulting in coma and death.

TREATMENT.

The best mode of procedure is to treat the special symptoms as they arise. The patient should be quarantined, placed in barracks, or large well-ventilated apartments, at a temperature not below 60° F., and where *plenty of fresh air* can be obtained. If the temperature rise to 107° or 108°, apply cold to the surface, and give antipyretic doses of *quinine*. Should the headache be severe, and the face flushed, *ice-bags* applied to the head may wonderfully relieve the patient. For vomiting, let him drink iced carbonic acid water, and should it be attended with restlessness and delirium, *morphine*, administered *hypodermically*, is indicated. When there is depression, *stimulants* must be given. If the mouth and pharynx become involved, ice, cold carbonic acid water, may be given, and use a solution of *muriated tinct. of iron*, or *carbolic acid*, or *permanganate of potash*, as a gargle.

In order to prevent pitting, cold applications, and rupturing each vesicle before it becomes a pustule, has been found beneficial.

As a preventive to the disease, vaccination should be performed with bovine virus or lymph.

SCARLET FEVER.

DEFINITION.

Is an inflammation affecting the tegumentary investment of the entire body, both cutaneous and mucous, and accompanied by fever of an infectious or contagious character.

VARIETIES.

Scarlatina simplex; scarlatina anginosa; scarlatina maligna.

MORBID ANATOMY.

The lesions occur in the skin and mucous membranes.

The eruption appears on the second and third days of the fever;

it consists of closely aggregated points about the size of a pin's head, with normal skin between these aggregations. The red spots are circular, and tend to become confluent. The skin may be turgid and swollen. The color changes with the fever, showing, when very red, that there is a high fever. The redness will, during the first forty-eight hours, if respiration be unimpeded, disappear under firm pressure, and reappear almost directly. The eruption may only appear on the face. The period of desquamation follows the period of decline in two or three days, and is due to an excessive production of newly-formed epidermis. The most frequent change in connection with those of the cutaneous surface is catarrhal pharyngitis. The tonsils become red, swollen, of a dry appearance, and covered with tenacious mucus. These changes may disappear in a few days, or become a dark livid color, and œdematous (associated with dysphagia, from œdema); inflammation of the parotids and other glands may also occur, often terminating in suppuration, or diffused necrosis, or gangrene of the tonsils, followed by abscesses, destroying the cell-tissues and sloughing of the skin in that region, and fatal hemorrhage.

This disease, in the early stages, is often complicated by diphtheria. During the eruptive stage, the ear (middle ear), as well as the throat, may be greatly affected. The kidneys are more affected in this disease than any other organ; catarrh of the uriniferous tubules, or croupous inflammation of the same in the cortical substance, commencing in the Malpighian tufts, may be developed. In a well-marked case of scarlatina nephritis, the epithelial cells of the tubules will be clouded, enlarged, changed in shape and position, and destroyed. The kidney may have the appearance of an interstitial nephritis. On *post-mortem* examination, there is found to be congestion of the brain, liver, spleen, etc., and softening of the liver and spleen. The mucous membrane of the intestines has become affected, and there is the "shaven beard" appearance in Peyer's patches.

SYNOPSIS OF THE FOREGOING.

The pathological lesions are confined to the skin, mucous membrane, organs, and blood.

In the skin—you may have the characteristic eruption before death, and evidences of desquamation in patches, or of local abscesses.

In the mucous membrane—there may be suppuration in the vicinity of the throat; ulceration of the tonsils, Eustachian tube, middle ear, and possibly of the cornea.

In the organs—desquamative nephritis, and evidence of some of the possible complications, such as pneumonia, pleurisy, peri- and endo-carditis, and neurosis of the middle ear.

In the blood—all the common lesions (see page 316).

ETIOLOGY.

This is a frequent disease of childhood, but may occur at any age. It is of a contagious nature, and may be conveyed directly from the affected to the healthy by contact; also through the atmosphere, or by clothing which has become saturated with the poison. It is apt to attack any or every member of a family, and is most infectious at the period of desquamation. The period of incubation is from two to ten days, but generally from four to seven days. Its regular course is from two to three weeks.

STAGES.

Stage of invasion—eruption—desquamation.

SYMPTOMS.

Stage of Invasion—(from twelve hours to four to five days).

Chilliness and slight rigor.
Headache.
Temperature—elevated, 103°–104°.
Pulse—accelerated, 120–130.
Face—flushed, dry, and red.
Throat—sore.
Neck—stiff.
Joints—tender.
Vomiting—projectile in character.
Thirst.
Tongue—red, and strawberry color.

Lassitude and pain in the back and limbs.
Restlessness.
Convulsions—in the very young or in the aged.
Delirium—in the very young or in the aged.
Coma—in the very young or in the aged.

Stage of Eruption.

Eruption—comes on twenty-four hours after the fever of invasion, and is of a *bright rose color*, if the attack be mild; or *deep red* (boiled lobster) color, if the attack be severe; it appears, first, on the neck and upper part of the chest and coalesces, extending over the body, face, and extremities.
On the fourth day, if you draw your finger across the surface, a well-defined line remains (sure sign).
Eruption remains for five or six days, beginning to fade on the fourth day, and disappears on the sixth day, when
There is a decrease in all the symptoms.
Urine—scanty, suppressed, high-colored and casts.
Blueness of the finger ends (showing defective oxygenation of the blood).
Œdema of the tonsils, uvula, and posterior walls of the pharynx, or ulcerative pharyngitis.

Stage of Desquamation.

Desquamation commences and the fever subsides.

IRREGULARITIES.

The most frequent are
Overwhelming of the cerebro-spinal system, which shows itself in convulsions, stupor, delirium, restlessness, blueness of the finger ends, picking at the bed-clothes, etc.
Œdema of the throat.
Temperature—excessive, may rise to 107° or 108°.
Pulse—greatly accelerated: may reach 140.
Interference with the return circulation.
Typhoid symptoms may occur from septic poisoning, and an ichorous discharge from the nose, indicating a sloughing pharyngitis (which is a very unfavorable sign).

Ulcers of the mouth.

Inflammation of the middle ear—producing delirium, intense pain, and rolling of the head (indicating acute meningitis).

Œdema glottidis.

COMPLICATIONS AND SEQUELÆ.

Scarlatina nephritis may develop, resulting in anasarca about the time of convalescence, showing itself first on the face. This condition commencing with restlessness, nausea, vomiting, then pain in the head, anorexia, photophobia, insomnia, and elevated temperature, followed by stupor: the urine becomes scanty and high-colored, containing casts of the exudative variety, or blood casts and albumen, or epithelium, denoting inflammation of the uriniferous tubules, convulsions, coma, and death, may terminate the case.

Other complications may occur, such as bronchitis; pneumonia; inflammation of the serous membranes; ulcerative endo-carditis; rheumatism; suppurative inflammation of the joints; diphtheria; suppuration of the middle ear; uræmia; and anasarca.

CAUSES OF DEATH.

Death may result from pyæmia; septicæmia; pneumonia; phthisis; or general anasarca.

DIFFERENTIAL DIAGNOSIS.

From measles; small-pox; erythema of surgical diseases; roseola, in this the anterior part of the pharynx is affected, and the symptoms are milder in character. In scarlet fever, the posterior part of the pharynx is affected.

PROGNOSIS.

Is uncertain. It depends upon the prevailing epidemics.

If the symptoms of the eruption appear within forty-eight hours; if it reaches its minimum on the second day; if the throat symptoms are mild; if slight dysphagia and slight glandular enlargements exist; if the temperature be not higher than 104°; pulse, 120; if the cerebral symptoms are not severe or of long dura-

tion, and a steady decline of temperature occur, with disappearance of the eruption, the prognosis is favorable, even if slight nephritic symptoms have been present, as they will disappear in from four to six weeks.

Should the temperature reach 105°, with extreme frequency of the pulse, dyspnœa, cold surface, small pulse, eruption of a livid hue, hemorrhage of the skin, suppurative pharyngitis (especially if extending to the nasal passages), copious coryza, nervousness, with typhoid symptoms, persistent vomiting, diarrhœa, nephritic symptoms, dropsy, complete suppression of urine, and the temperature inclined to rise, the prognosis is bad.

Is bad, if occurring from infancy to five years of age; in pregnancy; in organic diseases; or when complications exist.

TREATMENT.

Is both *preventive* and *medicinal.*

Preventive.—Strict quarantine is absolutely necessary, and the same precautions relative to the sick-room as in typhoid fever. The patient should have *plenty of fresh air* and free ventilation. Every article that may carry the infection must be thoroughly disinfected. Frequent sponging and rubbing the body over with oil after each sponging will prove a great comfort to the patient during the period of desquamation. *He must not be allowed to go out till desquamation is completed*, which usually takes three weeks after desquamation has commenced. After recovery has taken place, the whole of the premises, and every article that has been in use, must be scrupulously disinfected.

Medicinal.—Your duty is to stand by and watch and guard against complications as they arise. The bedding and body linen should be frequently changed. A warm bath should be given once a day, and the body well washed with carbolized soap when desquamation sets in, as it aids desquamation, and the kidneys are thus relieved. Never allow the temperature to rise to 104° more than twenty-four hours without reducing it by cold sponge or tepid saline baths, and also administer large doses of *quinine* (see typhoid fever, page 270). Do not use the cold bath unless the temperature rise to 105°.

For throat complications.—In the early stage, give cold carbonic acid water as a beverage, or allow ice to be held in the mouth. In the advanced stage, cloths wrung out in tepid or hot water may be applied to the throat. Warm water gargles and inhalations may be used with great benefit at the same time. It is better to use hot rather than cold applications in every case; but, whichever plan is adopted, *use either cold* internally and externally, or *hot* internally and externally. Should there be ulcerations in the throat, spray them with *carbolic acid*, *muriated tincture of iron*, *chlorate of potash*, *tannic acid*, or any of that class of remedies. Should there be great pain, *bromide of potassium* or *ether* used in the same way will afford relief. *Stimulants* may be resorted to early should there be feeble heart's action.

For kidney sequelæ.—Dry or wet cups, followed by hot fomentations, should be applied in the lumbar region, the temperature of the sick-room raised to 73°, and the same treatment adopted as in nephritis; or small doses of *calomel* may be given with a diuretic, and *water should be drank freely.* Should convulsions occur, *opium* should be administered either *hypodermically* or by the mouth. In this disease, symptoms of Bright's disease are now detected by the sphygmograph, showing increased arterial tension and development of uræmia.

MEASLES.

MORBID ANATOMY.

The blood is dark-colored, fluid, and poor in fibrin. The red corpuscles are diminished, and the white increased. There is a tendency to congestion of the internal organs. The liver and spleen are enlarged. There may be capillary bronchitis and pneumonia, which should be considered as part of the catarrhal affection of the respiratory organs. The eruption at first is papular, showing especially on the chin and gradually extending over the body; it is of a bright-red color, sometimes shading off into blue, disappearing on pressure, and returning again immediately. When the spots assume a dark-red color and do not disappear on pressure, capillary hemorrhages have taken place into the papules. The spots entirely disappear in from one to five days, leaving a yellow or brown stain behind which is due to pigmentation.

SYNOPSIS OF THE FOREGOING.

The principal lesions are in the skin, organs, and blood.

In the skin.—You have the eruption and a bran-like desquamation. There is catarrhal inflammation of the conjunctiva and mucous membrane of the respiratory passages; and there may be black or hemorrhagic spots on the skin.

In the organs.—Some of the complications which are possible, such as capillary bronchitis, pneumonia, phthisis, gangrene, and abscess of the lung. Otorrhœa and effusion into the serous cavities may be present.

In the blood.—All the common lesions of fevers (page 316) will exist.

ETIOLOGY.

It is a contagious and portable disease, and more easily contracted than small-pox or scarlet fever. The period of incubation is eight days. It may be induced by slight or prolonged exposure even with patients in the incubative stage.

STAGES.

Premonitory. Eruptive. And desquamative.

SYMPTOMS.

Premonitory stage.

Coryza.
Languor.
Chill (may be very indistinct).
Convulsions in children.
Eyes—suffused and red.
Photophobia.
Sneezing and pain over the frontal sinuses.
Bronchial catarrh.
Cough—dry, hoarse (iron cough).
Fever and catarrh—which last about forty-eight hours.

Eruptive stage.

Eruption—from *bright rose-red to mahogany*, and, on fading, becomes yellowish-red; appears first on the chin and face, then on the legs and arms, and lastly on the back of the hands; it takes four days to spread, and disappears about the sixth day. There are little red dots which are confluent, crescentic, and papillary, with swelling, itching, and a burning sensation.

Desquamative stage.

Desquamation—occurs in fine dust-like flakes. At this period the pulse is about 120 to 140, as a rule; the temperature usually about 103° (it rarely rises to 106°–107°).

DIFFERENTIAL DIAGNOSIS.

From scarlet fever and roseola. The catarrhal symptoms are the most prominent means of diagnosis in this disease.

From typhus (in children) when the mucous membrane of the pharynx settles it; as it is more injected than in typhus.

PROGNOSIS.

If there be no complications, the prognosis is good.

If dentition be progressing, it may render the prognosis bad.

If the hemorrhagic ulceration be of the typhoid variety (black measles), the prognosis is grave.

If it occur in pregnancy, or when there is profuse hemorrhage, the prognosis is bad; and in chronic diseases it is unfavorable.

Death usually occurs in the second week.

TREATMENT.

The patient must be quarantined, and kept in a large, well-ventilated room at a temperature of about 63°–65° F., and darkened so that the eyes may not be exposed to the light. The diet must consist of *milk*. Should there be itching and burning, sponge the body over with tepid water, as it aids in reducing the temperature. Convulsions may occur and cause death. *Stimulants* should never be used unless there be great prostration and bronchial affections. The utmost cleanliness must be observed. If the temperature rise to 103°–104° it must be reduced by tepid or cold sponging, and *quinine*. If the thirst be great, cold water may be given in small quantities as a beverage. If posterior pharyngeal catarrh, capillary bronchitis, or croupous laryngitis occur, *inhalations of steam* will be found very beneficial in relieving the symptoms. Should there be catarrhal pneumonia, *stimulants* must be given with judgment. If pulmonary complications arise, the patient must be protected from exposure. If there be great restlessness, give *pulv. Doveri* (small). If typhoid or hemorrhagic symptoms occur, use *stimulants* early.

RÖTHELN.

This disease may be confounded with measles and scarlet fever.

STAGES.

Invasion. Eruption. Desquamation.

SYMPTOMS.

Stage of Invasion.

Shivering.

Nausea and vomiting.

Sore throat.

Weakness.

Premonitory fever—which is short, and relieved when the eruption appears.

Stage of Eruption.

Eruption—appears simultaneously over the whole body; is sudden, less marked on the limbs than on the trunk, and fades in about four days without desquamation. On the first day, the eruption is like measles (minute dots); on the second day, irregular patches occur, varying from the size of a three-penny piece to that of a shilling; and on the third day, these are elevated and dark-red in the centre.

Temperature—is highest on the first day, never exceeding 102°; it falls on the second day to 100°; and on the fifth days becomes normal.

Stage of Desquamation.

Desquamation only occurs in rare cases; it resembles bran, and commences in the centre of the patch.

SEQUELÆ.

Swelling of the lymphatic glands; in rare cases, suppuration and dropsy.

PROGNOSIS.

Is favorable.

TREATMENT.

Avoid exposure to cold draughts of air, and protect the body with flannel. The medicinal treatment must be indicated by the symptoms which are most prominent.

TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN

TYPHUS.	TYPHOID.
CONTAGION.	**CONTAGION.**
Is contagious, infectious, and not portable.	Is non-contagious, but infectious. It requires prolonged exposure, gaining access to the body, through the air we breathe and the water we drink, thus becoming contagious.
SYNONYMS.	**SYNONYMS.**
Ship; jail; camp; putrid; hospital; fourteen day; petechial; cerebral typhus; exanthematous and continued fever.	Autumnal; abdominal typhus; enteric; gastro-enteric; gastric; forty day; dothentherica; ilio-typhus; and typhoid affection of Louis.
MORBID ANATOMY.	**MORBID ANATOMY.**
The principal changes will be found in the skin, organs, and blood.	The changes are confined to the skin, organs, and blood.
SKIN.—*Mulberry rash* present on the sides of the chest and abdomen, and possibly on the extremities. It disappears on pressure (for three days), and the penumbra is dark in color and somewhat elevated.	SKIN.—The eruption is confined to the abdomen, but it may appear on the chest, consisting of crops lasting three days each; they are few in number, slightly elevated, and called the *rose rash.*
BRAIN.—Hyperæmia of the vessels of the convexity. Meningeal exudations. Serous effusion of the pia mater and into the ventricles. The arachnoid is opaque and dotted with yellowish spots.	ORGANS.—The pathognomonic lesion is confined to Peyer's patches, the solitary and mesenteric glands, which undergo the following changes: 1. Congestion and the "shaven-beard" appearance. 2. Hyperplasia and hardening. 3. Softening of the glands from fatty degeneration. 4. Absorption or rupture of glands and evacuation of contents through sloughing or suppuration; which may result in perforation or formation of typhoid ulcers. The ulcer having its long axis parallel to the long axis of the intestine. 5. Cicatrices, etc., may form.
ALIMENTARY CANAL.—The changes are principally confined to Peyer's patches and the solitary glands which are found to be congested, and to have the "shaven beard" appearance.	BRAIN.—Hyperæmia of vessels, œdema, and punctate extravasations and appearance.
The evidences of *complications* are: bronchitis, pneumonia, splenization, pleurisy, œdema, meningitis, parenchymatous nephritis, ulceration of the larynx, pharynx, and mouth, and enlarged cervical glands	The evidences of complications are intestinal hemorrhage, peritonitis from perforation, pneumonia, parenchymatous nephritis, splenization of the lung, and catarrhal inflammation of the bronchi.
*BLOOD.—Common lesions.	*BLOOD.—Common lesions.

*N. B.—There are certain lesions common to all fevers – such as decrease in blood-corpuscles; staining of the surrounding tissues with hæmatin; soften-

THE FIVE PRINCIPAL ERUPTIVE FEVERS.

SCARLET.

CONTAGION.

Is contagious, infectious, and portable.

SYNONYMS.

Scarlatina simplex; scarlatina anginosa; scarlatina maligna, and red rash.

MORBID ANATOMY.

The changes are confined to the skin, mucous membranes, organs, and blood.

SKIN. — There are evidences of a *peely* desquamation, local abscesses, and characteristic eruption before death.

MUCOUS MEMBRANE. — Suppuration in the vicinity of the throat; ulceration of the tonsils, Eustáchian tube, middle ear, and possibly of the corneæ.

ORGANS. — Desquamative nephritis, and some of the possible complications, as pneumonia, pleurisy, peri-carditis, endo-carditis, and necrosis of the middle ear.

*BLOOD. — Common lesions.

MEASLES.

CONTAGION.

Is contagious, infectious, and portable.

SYNONYMS.

Rubeola.

MORBID ANATOMY.

The changes are in the skin, organs and blood.

SKIN.—There will be the characteristic eruption and *bran-like* desquamation; catarrhal inflammation of the conjunctiva, and mucous membrane of the respiratory passages, and black or hemorrhagic measles on the skin.

ORGANS. — Some of the complications possible, as capillary bronchitis, pneumonia, phthisis, gangrene, otorrhœa, abscess, and effusion in the serous cavities.

*BLOOD. — Common lesions.

SMALL-POX.

CONTAGION.

Is contagious, infectious, and portable.

SYNONYMS.

Variola discreta; variola confluens; variola hemorrhagica, and varioloid.

MORBID ANATOMY.

The changes are in the skin, mucous membranes, organs, and blood.

SKIN.—All the stages of eruption which may be macule, papule, vesicle, pustule, scab, or cicatrix; also, possible abscess, ulceration and local slough.

MUCOUS MEMBRANE. — Within the urethra, eye, nose, mouth, and middle ear, the same conditions are possible.

ORGANS. — Parenchymatous nephritis, œdema glottidis. Evidences of complication, as pleurisy, pneumonia, peri-carditis, endo-carditis, meningitis, bronchitis, and fluid blood, may be found in some of the serous cavities.

*BLOOD.—Common lesions.

the fibrin and albumen of the blood, disorganization and serration of the red
ing of the mucous membrane of the alimentary canal and the various organs.

TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN

TYPHUS.	TYPHOID.
ETIOLOGY.	ETIOLOGY.
Is epidemic, and produced by some animal poison arising from human effluvia; from overcrowding, and filth; is communicable by direct contact.	Is endemic, and due to decomposing organic matter, mixed with typhoid material.
STAGES.	STAGES.
Incubation, invasion, and eruption.	Incubation, invasion, and eruption.
ADVENT.	ADVENT.
Suddenly, with intense chill and marked symptoms, and steady increasing headache, delirium and great prostration.	*Insidiously*, with general malaise, vomiting, headache, photophobia; chill is rare; five days elapse before patient takes to his bed.
DURATION.	DURATION.
Fourteen days.	Twenty-one to forty days.
CRISIS.	CRISIS.
On twelfth day, accompanied by a sudden termination of symptoms and a healthy sleep.	None. Tympanites gradually disappearing, and the tongue gradually clearing.
TEMPERATURE.	TEMPERATURE.
Very high; on the second day 104°; frequently 105° to 107°, about third day; remains so till convalescence. Is a higher temperature than any other affection but sunstroke and bilious remittent and relapsing fever.	Very little increased at onset. Is exacerbating, seldom above 104° or 105°, and reaches its maximum about the seventh day; begins to fall about the end of third week, and becomes normal at the end of the fourth week.
PULSE.	PULSE.
First week about 110 to 120. Second week generally 100, frequent, soft, rapid, and easily compressible. It may be irregular.	During first week 98 to 110. In second week falls, or may even rise to 140. Third week is variable. Fourth week falls to about normal.

THE FIVE PRINCIPAL ERUPTIVE FEVERS.

SCARLET.

ETIOLOGY.

Contagion direct and by the clothes. It may go through a whole family. Is most infectious at the time of desquamation.

STAGES.

Incubation, invasion, eruption, and desquamation.

ADVENT.

Period of incubation is from two to ten days, but generally four to seven. Commences in children with headache, vomiting, general malaise, rash, and sore throat.

DURATION.

Twenty-one to twenty-eight days.

CRISIS.

About the fourteenth day. (?)

TEMPERATURE.

May reach 105° or 107°; is higher during the eruption, and begins to subside about the tenth day. It has no secondary fever.

PULSE.

Accelerated; may even reach 140.

MEASLES.

ETIOLOGY.

Is a portable disease, and more tenacious than scarlet fever and small-pox.

STAGES.

Premonitory or precursory; eruptive and desquamative.

ADVENT.

Period of incubation is eight days.

Commences with chill, coryza, frontal headache, lassitude, harsh cough and fever, which usually lasts about forty-eight hours.

DURATION.

Fourteen to twenty-one days.

CRISIS.

None.

TEMPERATURE.

May reach 105°, and sinks to normal twenty-four hours after appearance of the rash. It has no secondary fever.

PULSE.

Slightly accelerated.

SMALL-POX.

ETIOLOGY.

Is propagated by contagion; by the breath and exhalations from the skin (at two and a half feet distant in the open air); can be conveyed by clothing, etc., and attack all ages.

Is more infectious during the stage of suppuration or diminution, or even incubation.

STAGES.

Incubation; invasion; eruption; suppuration; desiccation.

ADVENT.

Period of incubation is from ten to thirteen days. If from inoculation (48 hours). It commences with an initiatory stage of chilliness, severe pain in the back, loins, head, and nausea, etc.

DURATION.

Four to five weeks.

CRISIS.

About twenty-first day. (?)

TEMPERATURE.

Is often 106° at onset, and may reach 109°. After eruption appears, it usually falls to 100°.

Secondary fever very high on the eighth day, and falls slowly.

PULSE.

May rise from 100 to 120, or even 140.

TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN

TYPHUS.	TYPHOID.
ERUPTION.	ERUPTION.
Small, irregular-shaped, brick dust spots, slightly elevated at first, called the "*mulberry rash.*" Remains throughout the disease. Appears on the fifth to the seventh day of the fever, and disappears on pressure for the first two days of the eruption. Appears chiefly on the sides of the chest and extremities.	Lenticular spots, slightly elevated. *Bright rose* color. Appears in successive crops (each crop lasting three days), and disappears, when another crop takes their place. It disappears on the *slightest* pressure, and *reappears instantly.* Appears on the abdomen chiefly about the seventh day.
CEREBRAL SYMPTOMS.	CEREBRAL SYMPTOMS.
Active delirium and terrific headache from the onset.	Are not present till about the second or third week, and appear as a muttering delirium.
CONDITION OF THE PUPILS.	CONDITION OF THE PUPILS.
Contracted.	Dilated.
EPISTAXIS.	EPISTAXIS.
Rare, and of no importance.	A frequent and grave symptom, if appearing late in the disease.
CONDITION OF TONGUE.	CONDITION OF TONGUE.
First, swollen and white-coated; after a day or two, becomes of a yellowish-brown color and thick; later on, dark, dry, and fissured.	Is first of a light white color; then red on the tip and sides, and dry in in the centre; then heavily coated with sordes on the teeth and mouth, and, after a time, becomes clear and shining, like beef.
COUNTENANCE.	COUNTENANCE.
Dull and heavy, and later on of a *mahogany* color (drunken face).	Pale, *olive*, or *leaden* look.
DESQUAMATION.	DESQUAMATION.
None.	None.

THE FIVE PRINCIPAL ERUPTIVE FEVERS.

SCARLET.	MEASLES.	SMALL-POX.
ERUPTION.	ERUPTION.	ERUPTION.
Appears 24 hours after the fever of invasion. If the attack be mild, the color is *bright rose;* but if severe, it will be a *deep-red lobster color.* If the fingernail be drawn over the eruption, a white line will remain, which will last for two or three minutes. It appears first on the neck, and spreads rapidly.	It appears on the fourth day as little dots, *like flea bites,* of a *rose-red* or *mahogany* color. May be confluent, crescentic, and papular. If the fingernail be drawn over the eruption, a line appears, which disappears as quickly as the pressure is removed. It appears first on the face, round the mouth, and on the neck, then on the legs and arms, and lastly, on the back of the hands.	Appears about the third or fourth day. 1st. As a macule. 2d. Papule. 3d. Vesicle. 4th. Vesicle becomes umbilicated. 5th. Vesicle changes color, and becomes a pustule, which may slough, and result in cicatrix and pitting; or 6th. Scab. It appears first at the root of the hair, lips, palate or fauces.
CEREBRAL SYMPTOMS.	CEREBRAL SYMPTOMS.	CEREBRAL SYMPTOMS.
Frequent and grave.	Usually absent.	Frequent; delirium probable about the third day. Convulsions in children.
CONDITION OF THE PUPILS.	CONDITION OF THE PUPILS.	CONDITION OF THE PUPILS.
May be contracted.	Normal.	May be contracted.
EPISTAXIS.	EPISTAXIS.	EPISTAXIS.
Absent.	Absent.	Infrequent.
CONDITION OF THE TONGUE.	CONDITION OF THE TONGUE.	CONDITION OF THE TONGUE.
"Strawberry" tongue.	Coated, and red on the edges. Throat is sore; coryza; and bronchitis.	Coated and swollen, and red on the edges. Sore throat and dry cough.
COUNTENANCE.	COUNTENANCE.	COUNTENANCE.
Flushed, dry, and red, and headache.	Suffusion and redness of the eyes, and coryza.	Anxious and flushed; photophobia and headache.
DESQUAMATION.	DESQUAMATION.	DESQUAMATION.
Occurs in large patches, especially from the hands and feet, with itching, and successive desquamation (*peely*) may occur.	Is bran-like, and has an odor of mouldy cheese (*mealy*).	Scabs, crusts, and thick scales in large quantities, with *intolerable* itching (*scaly*).

TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN

TYPHUS.	TYPHOID.
EMACIATION.	EMACIATION.
Slight.	Great.
CONDITION OF THE BOWELS.	CONDITION OF THE BOWELS.
Constipated; no tympanites nor tenderness.	Diarrhœa (*pea soup*). Tympanites.—This is a bad sign; it indicates gas in the intestines. Pain and gurgling in the right iliac fossa.
INTESTINAL HEMORRHAGE.	INTESTINAL HEMORRHAGE.
Rare.	Frequent.
CHANGES IN URINE.	CHANGES IN URINE.
Is first diminished, then increased; and suppression may occur later on in the disease. There are vesical, renal, and fatty casts; and albumen and urea are increased.	Increased in quantity. No albumen; casts are rare. Urea is diminished at first, and increased later on.
COMPLICATIONS.	COMPLICATIONS.
Subacute meningitis; pulmonary gangrene; capillary bronchitis; pneumonia; pleurisy; congestion; œdema; laryngitis; and Bright's.	Peritonitis; abscesses; pleurisy, with pus exudation; simple bronchitis; catarrhal pneumonia.
SEQUELÆ.	SEQUELÆ.
None.	None.
CAUSES OF DEATH.	CAUSES OF DEATH.
Coma; syncope; capillary bronchitis; gangrene of the lung; meningitis; pneumonia; exhaustion, from the effects of the poison; Bright's granular kidney.	Toxæmia; debility; suppression of the excretory function of the kidney; hyperæmia and œdema of the lungs; intestinal hemorrhage; exhaustive diarrhœa; intestinal perforation; peritonitis, with or without perforation; relapse from absorption of the poison; cathartics, if given during the first week.

THE FIVE PRINCIPAL ERUPTIVE FEVERS.

SCARLET.	MEASLES.	SMALL-POX.
EMACIATION.	EMACIATION.	EMACIATION.
Slight.	Not marked.	Great.
CONDITION OF THE BOWELS.	CONDITION OF THE BOWELS.	CONDITION OF THE BOWELS.
Not specially affected.	Not specially affected.	Not specially affected.
INTESTINAL HEMORRHAGE.	INTESTINAL HEMORRHAGE.	INTESTINAL HEMORRHAGE.
Absent.	Absent.	Absent.
CHANGES IN URINE.	CHANGES IN URINE.	CHANGES IN URINE.
In the latter stages, urine becomes scanty, high-colored; contains casts, as epithelial, small hyaline, blood, and blood-globules; indicating the inflammatory stage of parenchymatous nephritis.	None.	May be scanty and high-colored, and contain casts at the commencement of the stage of suppuration; indicating the first stage of parenchymatous nephritis.
COMPLICATIONS.	COMPLICATIONS.	COMPLICATIONS.
Pleurisy (frequent); bronchitis; pneumonia (in rare cases); inflammation of serous membranes; œdema glottidis; ulcerative endocarditis and diphtheria.	Pneumonia (frequent); lobar pneumonia; phthisis; ophthalmia; capillary bronchitis; abscess of the lung; gangrene of the lung; Bright's; gastric catarrh; profuse hemorrhage.	Pneumonia (in rare cases); bronchitis (general); œdema glottidis.
SEQUELÆ.	SEQUELÆ.	SEQUELÆ.
Bright's; dropsy; deafness; conjunctivitis; phthisis; chronic diarrhœa; and glandular enlargements.	Chronic bronchitis; conjunctivitis; phthisis; otorrhœa.	Chronic diarrhœa; abscesses; glandular enlargements; various diseases of the eyeball and eyelid.
CAUSES OF DEATH.	CAUSES OF DEATH.	CAUSES OF DEATH.
Convulsions, from excessive blood-poison; bronchitis; pneumonia; pyæmia; diphtheria; septicæmia; phthisis; general anasarca; and Bright's.	Any of the above *complications*, especially capillary bronchitis in children, and phthisis in the adult.	*Œdema glottidis;* general bronchitis; pneumonia; pyæmia; serous inflammation of the brain and heart. Acute fatty degeneration of the kidney.

INFARCTIONS.

DEFINITION.

Are wedge-shaped spots of discoloration and consolidation in an organ; dependent upon capillary obstruction and collateral hyperæmia. Their base usually looks toward the periphery of the organ in which it occurs, and the apex toward the centre.

VARIETIES.

1st. *Thrombic*—in which the veins, where the seat of stoppage occurs, are distended and engorged. The obstruction, in this form, is dependent upon a mechanical or inflammatory stasis.

If a vein be plugged by a thrombus, the arteries rupture from collateral circulation.

2d. *Embolic*—the obstruction usually takes place in the arteries. A clot is first formed in a vein, and passing onward to the heart, is forced into the lung, or other organs, and plugs some artery of those organs. It may be due to a vegetation from the valves of the right heart.

If an artery be plugged by an embolus, the veins usually rupture from collateral circulation.

3d. *Pyœmic*—which may be either one of the above-named varieties, for explanation see pages 326 and 327. This type of infarction usually suppurates; as the obstruction in the vessels, if due to thrombosis, depends on a poisoned condition of the blood; while, if due to an embolus, it depends upon a suppurative phlebitis of some point more or less remote from the lungs, which has caused a disintegration of a blood-clot (thus causing emboli).

SEPTICÆMIA.

DEFINITION.

Is a blood-condition dependent upon absorption of decomposing animal matter by the lymphatics, when placed in actual contact

with a living membrane (example—decomposing placenta in the uterus).

MORBID ANATOMY.

The severity of the disease depends upon the amount of poison absorbed. It is a disease that may produce death in twenty-four hours if a large quantity of the poison has been introduced into the system, or it may become a continued fever when small quantities are gradually introduced. It may be recovered from, in some cases, by arresting the development of the poison, or by the removal of the cause. The principal lesion in this disease occurs in the blood which will be found to lose its normal power of coagulating, is darker than normal, and undergoes decomposition when drawn from the body; but should coagulation take place, the coagula will be found to break down very easily. On *post-mortem* examination, the liver will usually be found congested and œdematous; the spleen and mucous membranes softened; the salivary glands enlarged; and the muscles of the heart weakened.

ETIOLOGY.

It can only occur when decomposing animal matter is brought into direct contact with the living membrane. It occurs in gangrene and abscess of the lung; suppurative endocarditis; retained placenta; dead fœtus in utero; post-mortem wounds; diphtheria; introduction of sulphuret of ammonia into the system; decomposing urine in the bladder; typhoid ulcers, etc.

SYMPTOMS.

Chill—but not distinct (rather rigors).

Sudden fever.

Temperature—(depends upon the amount of poison absorbed) in mild cases, 102°–103°; in severe cases, 105°–107°.

Pulse—may rise to 120 or 140.

Stupor—the patient answers questions stupidly.

Profuse perspiration.

Dinginess and dryness of the surface (following the profuse perspiration).

Typhoid symptoms.

Jactitations.
Delirium—low muttering; the patient probably passes on to coma.
Diarrhœa—profuse watery (is an effort of elimination on the part of Nature).

DIFFERENTIAL DIAGNOSIS.

From pyæmia—from which it differs in not resulting in metastatic abscesses; although there are chills, still they are not recurrent as in pyæmia.

PROGNOSIS.

Depends upon the amount of poison present in the system. It is fatal when the poison is intense.

TREATMENT.

Same as in pyæmia (see page 329).

PYÆMIA.

DEFINITION.

Is produced by a miasm, generated by decomposing pus, and characterized during life by chills (recurring), fevers, and sweats, and, after death, by metastatic abscesses all over the body.

MORBID ANATOMY.

There is a peculiar jaundiced appearance to the skin, which is characteristic of the disease. The blood, as a result of the miasmatic infection, exhibits a tendency to coagulate spontaneously, especially where the current is slowed, as in the capillary vessels. Fine coagula will be found in the heart, also an interlacing of the chordæ tendineæ. In the various organs throughout the body, there will be infarctions, which result in abscesses. The mucous membranes of the body will be found to be thickened.

ETIOLOGY.

It is often due to a suppurative capillary phlebitis, occurring in persons suffering from disordered or low conditions of the blood. It may be either local or general; should it be local, the symptoms

will be rapid, but if general, it will take some time before thorough development of the symptoms takes place. It may occur in either closed or open wounds in the following manner:

In closed wounds—(1) from a blow (say on the head); (2) phlebitis of the diploë ensues; (3) a clot is formed in the veins at the seat of the injury; (4) suppuration of the clot and breaking down of the same takes place (this causes loosening of the clot, whose particles pass through the large blood-vessels to the heart, and thence to the lungs), resulting in (5) an embolus of the lung or other organs, which causes (6) an infarction. This condition is evidenced by a wedge-shaped spot of discoloration and consolidation, dependent on capillary obstruction and collateral hyperæmia; its base usually looks toward the periphery of the organ, and its apex toward the centre. The embolus having been a poisoned one, since it was due to unhealthy suppuration, the infarction undergoes a similar suppuration; its coagula break down and multiply emboli in the blood, which are carried through the left heart into the general circulation. It is in this manner that metastatic abscesses are formed all over the body, since these emboli become lodged in other organs, and then generate infarctions which again suppurate.

In open wounds.—Pyæmia is due (1) to some miasmatic atmospheric conditions (probably generated in decomposing pus); (2) the miasm is probably absorbed by the blood-vessels, and not by the lymphatics; (3) the effect of the miasm is shown by a tendency in the infected blood to *coagulate spontaneously in the small capillary vessels where the current is slow;* (4) the small coagula thus formed (thrombi) in different parts of the body create suppurative and metastatic abscesses, on account of their poisoned character. The duration of the disease is from eight to ten days, or it may even extend to three weeks.

SYMPTOMS.

Initial symptoms—are usually well marked.

Chills—which are sharp, long, and recur at intervals for several days.

Profuse sweating—which follows every chill.

Temperature—rises before the chill occurs to 104° or 105°. This rise either precedes or accompanies the chill, and also the perspiring stage; but, after the perspiring stage, the temperature may even become normal, as occurs in the puerperal type, or it may have a marked rise, 107°.

Pulse—rapid (140 to 200), small, feeble, and rarely irregular.

Restlessness.

Delirium—which will be of a low and muttering character.

Cellulitis—especially in the glands with loss of vitality of adjacent tissues and subsequent suppuration,

Exhaustion—extreme.

Jaundice—(of the hematogenous variety) due to the changes in the red globules of the blood from the action of the pyæmic poison, or to the action of the poison on the nerve-centres.

Respiration—hurried, especially just before a chill or sweat.

Breath—has a sweet, nauseating odor.

Perspiration—has a sweet, nauseating odor, staining the clothing to a dirty yellow color.

Urine—is at first scanty and high-colored, then becomes more abundant; the high color is due to changes in the coloring of the blood.

Diarrhœa—profuse watery.

Cough.

Expectoration—sometimes streaked with blood, or may be pneumonic.

Emaciation and feebleness.

Tongue—is at first glairy, and subsequently heavily coated with sordes, and further symptoms of a typhoid variety ensue.

DIFFERENTIAL DIAGNOSIS.

Is easily made. It may be confounded with some of the infectious fevers as septicæmia; but, in this latter case, you rarely have a second chill, and, if you do, it always follows the fever. There is also the peculiar hue to the skin to enable the diagnosis of pyæmia to be made. It may also be mistaken for acute articular rheumatism, or for those cases of scarlet fever when suppuration occurs.

PROGNOSIS.

Is worse than in almost any other disease.

TREATMENT.

The prophylactic treatment will only interest the surgeon, and also the physician, during the puerperal stage. When there is an unhealthy suppuration, antiseptic treatment will tend somewhat to stop it. Of all the curative remedies, *fresh air* stands first to overcome these diseases. Let the patients be placed in tents or pavilions, where they will have as free ventilation as in tents, and use all the means of disinfection. Diaphoretics and diuretics have been advised, but it was found that the patients went much more quickly into a state of collapse than when they were left alone. Sulphurous acid has been advised, but this was so difficult to administer, that *sulphites* (gr. xx.–xxx. doses) of *sodium*, *calcium*, and *magnesium* were administered, as they were claimed to neutralize the poison, but this treatment was not found by clinical experience to have the desired effect. *Quinine* has been suggested on account of its having such a wonderful effect in malarial diseases, and this, like its predecessors, has been found to be of no avail, and has also been abandoned. *Stimulants* should be administered *early* and *in large quantities*, and so keep up the patient's strength as much as possible. *Opium* should be given *hypodermically* to quiet the restlessness that occurs prior to the chills and sweatings. The patient's body, bed, and linen should be kept scrupulously clean. The diarrhœa and perspiration should not be checked. *Good nursing is indispensable*, and concentrated liquid food, as milk with lime-water, eggs, beef-tea, at short intervals.

ERYSIPELAS.

DEFINITION.

Is a diffuse erythematous inflammation of the skin or the mucous and serous membranes, which rapidly spreads, and is attended with febrile symptoms. It is both contagious and infectious.

VARIETIES.

(1) Cutaneous or simple; (2) phlegmonous; and (3) internal.

Cutaneous variety—attacks the superficial portion of the skin.

Phlegmonous variety—attacks the sub-cutaneous areolar tissue, as well as the cutaneous surfaces.

Internal variety—where the serous and mucous membranes are involved, producing cellulitis, also involving the fasciæ, aponeuroses, etc., etc.

ETIOLOGY.

Predisposing causes.—Bad hygiene; filth; deprivation of food, etc.; over-fatigue; over-anxiety; intemperance; sedentary habits; indolent life; high living; diabetes; albuminuria; drinking bad water, etc.

Exciting causes.—Change of temperature; cold or damp air; wounds.

SYMPTOMS.

Cutaneous variety.

Face—chiefly affected (which becomes red, œdematous, and distorted).
Chill.
Temperature—rises to 103°-104°.
Nausea.
Vomiting.
Stupor.
Eyes—closed by swelling and œdema.
Delirium.
Typhoid symptoms—which abate in about eight to twelve days.
Skin—grows pale, scaly; abscesses or boils may form.
The beard falls out and frequently returns.
This variety also occurs from wounds in the head.

Phlegmonous variety.

Skin—over the affected portion becomes tense, shining, and subsequently sloughs, and exhaustive suppuration ensues.
Duration depends upon the severity of the attack.

DIFFERENTIAL DIAGNOSIS.

From phlebitis—which gives the feeling of a hard cord running

across the inflamed part. It attacks the walls of the veins instead of the tissues and lymphatics, as in erysipelas.

PROGNOSIS.

The *cutaneous* or *simple variety* generally gets well, but it may attack some other organ, as the lungs, peritoneum, or pelvis, or even the brain. In the strong and healthy, the duration is from seven to fifteen days; the first four days it usually tends to spread; the second four days it gradually clears up, and it takes two days more to get well.

In the *phlegmonous variety*, the prognosis is unfavorable, as you have suppuration; and especially unfavorable if occurring in young children, and those addicted to intemperance and bad living.

TREATMENT.

Is both preventive and curative.

Preventive.—First remove all obstacles. Keep the patient in bed in a large, well-ventilated room; perfect cleanliness must be observed. The diet should be nutritious, and stimulants avoided, if possible.

Curative.—This is divided into *constitutional* and *local* treatment.

Constitutional.—Be on your guard against fever. The patients must be supported in moderation, should there be any signs of an asthenic (debilitating) fever, by giving *stimulants*, such as milk punches, egg-nog, port wine (by the bottle), strong beef-tea, bark, ammonia, etc. Large doses of *tr. ferri mur.*, gtt. xv.–℥ ss. every two or three hours day and night.

Local.—In the *simple form* the inflamed parts should be painted over with butter-milk; it cools and protects them from the air, or bathe the parts with the following lotion: ℞ plumbi acet., ℥ i.; opii, ℥ ss.; aquæ, O.i.; or a lotion made of ipecac and water. Do not use iodine, as it only irritates the parts, nor nitrate of silver, as it is of no service.

In the *phlegmonous form.*—Incisions should be freely resorted to. Do not be afraid of free incisions, as it greatly relieves the patient; afterward poultice the part with linseed meal. Keep the limb

well covered over with cotton wool, and so soon as the discharge is well carried away, and the parts assume a healthy appearance, commence the process of healing by strapping.

RHEUMATISM.

Is a disease dependent upon a morbid condition of the blood, due to a poison (probably lactic acid), which is developed within the body, having the power of rendering the secretions and excretions acid, and associated with an alteration in the various salts, favoring an increase in the fibrin of the blood.

VARIETIES.

Acute and chronic articular; deforming articular; muscular; and gonorrheal.

ACUTE ARTICULAR RHEUMATISM.

SYNONYM.

"Flying gout."

DEFINITION.

Is an inflammatory disturbance of nutrition, located in the synovial capsule of one or more of the joints of the body.

MORBID ANATOMY.

On *post-mortem* examination, the synovial capsule of the affected joints may be found to be of a dark-red color (due to hyperæmia or ecchymosis); it will also be relaxed and puffed up, and the joint dilated and filled with purulent fluid. The blood in the heart and large blood-vessels shows large deposits of fibrin. Heart lesions are frequently developed in the course of this disease.

ETIOLOGY.

It is a disease which occurs principally among the poorer classes (as in fishermen, etc.). It usually occurs between the ages of fif-

teen and forty years, from possibly an hereditary tendency, the mode of life, or a faulty digestive process. Its exciting causes are exposure to cold, by suddenly wetting the heated body; by exposure to a draught; or living in damp places. The duration of the disease is usually from two to six weeks.

SYMPTOMS.

Usually of sudden advent.
Chill—rare.
Fever.
Pain—in one or more of the joints, which usually increases, and is worse on motion.
Swollen and tender joints—the large joints are principally affected.
Skin over the joint—may be normal, of a light or dark-red color.
Temperature—usually 100°-101°; may rise to 104°-105°.
Pulse—full and soft, 90-100; may rise to 130.
Perspiration—peculiar acid.
Great thirst.
Urine—scanty, high sp. gr.; urea increased.

COMPLICATIONS.

Peri-carditis; endocarditis; myo-carditis; pleurisy; pneumonia; cerebral and spinal meningitis; and pyæmia, from suppuration.

CAUSES OF DEATH.

Death may occur from complications, or sudden collapse from an effect upon the nervous system.

DIFFERENTIAL DIAGNOSIS.

From synovitis; arthritis; and gout.

PROGNOSIS.

It usually terminates in complete recovery, provided there be no complications. Young subjects are very liable to have cardiac complications from endo-carditis and endarteritis.

TREATMENT.

The patient should live in a country where the atmosphere is dry, and with a temperature ranging about 70°; his body should be covered with flannel. The affected joints should be packed in a poultice made of *bicarb. sodii*, bound round with cotton wool, and covered with oil-silk. All animal food should be withheld, and the diet consist of milk with an alkali. *Lime juice* may be drank, or *lemons* eaten, with advantage, as in the body this acid is changed into an alkali. In some cases a hot pack may be found to greatly relieve the sufferings of the patient. Should there be great pain, *opium* may be given, and in the event of swelling, the parts may be painted with *iodine*. If meningeal troubles occur, *leeches* and *cold compresses* should be applied to the head. In a few cases, *salicylate of soda* will sometimes stop the disease. *Pulv. sodii bicarb.* and *pulv. acidi tartar.* may be given as an effervescing drink. The Germans advise giving *salicylate of soda* (gr. x.–xv.) every four hours, or *capsules of salicylic acid*, gr. x., every two hours, and crowding them continuously.

CHRONIC ARTICULAR RHEUMATISM.

MORBID ANATOMY.

The synovial capsule and ligaments of the joints will be found to be thickened; hypertrophy or degeneration of the membranes may exist; the cartilages are often relaxed and shaggy; and the synovial fluid cloudy. It may attack only one joint at a time, sometimes inducing comparatively little anatomical change. It may lead to suppuration of the joint, and caries of the end of the bone.

ETIOLOGY.

It may occur from congenital predisposition; from previous attacks; from catching cold; or remaining in cold, damp, windy places.

SYMPTOMS.

Pain—constant and increased, especially at night, on pressure or

active motion; or it may only occur at every change of the weather.

Crackling sensation or crepitation felt if the hand be placed on the joint when motion is made.

Swelling—(from increase of synovia, and thickening of the capsule and ligaments).

Prominence of the joint—which may be swollen and painful.

Fever—may occur, with frequent pulse, and profuse perspiration.

Urine—thick and sedimentary.

Emaciation.

It may be complicated with neuralgia and paralysis.

DIFFERENTIAL DIAGNOSIS.

From chronic gout.

PROGNOSIS.

This disease often ends in a permanent deformity.

TREATMENT.

Flannel should always be worn next to the body. *Iodide of potassium*, *guaiacum*, *oil of turpentine*, and *cod-liver oil* are largely used in this affection, but local treatment is found to give more relief. The inflamed part may be painted with the *tincture of iodine* or a lotion composed of tr. iodine, ℈ss.; pot. iodide, ʒ ss.; aquæ, ℥ iij. may be applied; or a liniment containing *oil of turpentine*, *oil of sassafras*, *ammonia*, and *laudanum* diluted with soap liniment; or when the pain is excessive, *chloroform* or *aconite* liniment may be used. Blistering may even be resorted to in bad cases. Where there is rigidity of the joints, and even pain in them, or, in the muscles, *hot water* may be constantly poured over the parts. Some prefer the hot water or *vapor bath* and others the *hot dry air* bath at a temperature of about 130° to 200°, as a means of affording great relief. In order to aid in the restoration of the stiffened parts, *electricity* should be resorted to.

MUSCULAR RHEUMATISM.

ETIOLOGY.

It is a rheumatism of the muscles, fascia, periosteum, and other fibrous tissues; the changes consisting chiefly of hyperæmia and scanty exudations. It is diffcult to say whether the sensory nerves traversing the muscles are morbidly excited by changes in the muscles and sarcolemma, or whether simultaneous changes occur in the neurolemma.

SYMPTOMS.

Pain—designated as stretching or tearing; it is increased by motion and cold applications; diminished by pressure and warm applications, and may be wandering or fixed.

Neck—may be stiffened (as its muscles are most often affected).

TREATMENT.

The parts should be painted with *iodine*, or a blister in some form. One of the most effective rubefacients is the application of the *induced current* by means of the electric brush. Vapor baths may prove of great relief to the patient. Should there be great pain, the *hypodermic* use of *morphia* can be resorted to.

DIABETES.

VARIETIES.

"Mellitus" and "Insipidus."

DIABETES MELLITUS.

SYNONYMS.

"Glycosuria;" "milleturia."

DEFINITION.

Is characterized by excessive and saccharine urine.

MORBID ANATOMY.

Is obscure. Physiologists claim, from experiments, the probable seat of the disorder is in the liver (due to its glycogenic function). It is also claimed that, by irritating the fourth ventricle of the brain, sugar can be produced in the urine. It is also regarded as a nervous disease, through the pneumo-gastric nerve; for if you cut the pneumo-gastric, no sugar will be found in the liver after death.

ETIOLOGY.

Predisposing causes.—It occurs more frequently in men than in women, especially the young and middle-aged adults. It may be hereditary, or occur from nervous shocks.

Exciting causes.—Exposure to wet or cold; drinking cold water in large quantities when the patient is heated; intemperance; fevers; injuries to the brain and spinal cord (through the pneumo-gastric).

SYMPTOMS.

Come on slowly with malaise.

Thirst—incessant (a prominent symptom).

Emaciation.

Insatiable hunger.

Excessive micturition.

Urine—pale straw color, sp. gr. 1030 to 1050. Patients have been known to pass seventy to eighty pints in twenty-four hours.

Anæmia.

Dyspeptic symptoms—due to faulty secretion of the gastric juice.

Palpitation of the heart.

Vertigo.

Tongue—is glazed and furrowed.

Mouth—clammy.

Failure of mental and sexual powers (the latter may be sometimes increased).

COMPLICATIONS.

Tubercular phthisis; furuncles and anthrax; gangrene of the lower extremities; defective vision; cataract; pruritus vulvæ; erysipelas, etc.

CAUSES OF DEATH.

Fever; œdema; diarrhœa; and probably œdema of the lungs, or over-working of the kidneys or some other organ may produce death.

PROGNOSIS.

Is unfavorable, although recovery is not impossible, usually lasts from one to three years.

TREATMENT.

The tests applied for sugar in the urine are principally Moore's Tromer's, and Fehling's. It is the weight of the patient that denotes improvement in the disease and not in the amount of sugar passed. Prohibit the use of sugar or starchy food, as bread (except bran bread), potatoes, cabbage, broccoli, celery, lettuce, spinach, and onions. Milk and liver should also be amongst the number. The patient should be fed on meat, eggs, butter, jellies, aërated bread, tea and coffee not sweetened. Stimulants must be avoided, but water may be used ad libitum.

Medicinal agents are of no use, excepting *opium*. *Codeia* especially. *Oleum morrhuæ* may be given to keep up the strength of the patient, or rub the surface of the body with oil. The radical cure for this disease has not yet been discovered.

DIABETES INSIPIDUS.

SYNONYM.

"Polyuria."

DEFINITION.

There is an excessive amount of urine passed, which is of a light color, light in weight, and contains neither sugar nor albumen, but is accompanied by excessive thirst (polydipsia).

MORBID ANATOMY.

Is obscure. There is generally atrophy of the kidney or renal congestion associated with this variety of diabetes. It is stated that the immediate cause of this disease is dilatation of the capillary vessels of the kidneys, due to disturbance of the ganglio-

nervous system which controls the circulation of that organ. The disease may last from a few weeks to many years, and may be congenital.

ETIOLOGY.

From blows on the head; exposure to cold; drinking cold fluids when the patient is heated; cerebral disease; intemperance.

SYMPTOMS.

Usually sudden.
Intense thirst.
Skin—dry and harsh.
Great emaciation and debility.
Excessive urination—about fifty to seventy pints (passed in a day).

TREATMENT.

No specific cure has yet been discovered. The only treatment is to hold the disease as much in check as possible. The following drugs have been suggested: *nitrate of potassium, valerian, ergot, iron, alum, lime-water, tannic and gallic acid, bromide of potassium,* etc. Also blistering the nape of the neck.

SCURVY.

SYNONYM.

"Scorbutus."

DEFINITION.

Is a peculiar form of anæmia, arising from deficiency of vegetable diet, attended with a tendency to the occurrence of hemorrhages, impairment of nutrition, and great mental and bodily prostration.

MORBID ANATOMY.

The body will be found to be greatly emaciated. Slight œdema will exist, especially of the lower extremities. Extravasations of blood may be found not only in the superficial regions of the body, but in the substance of the lungs, beneath the pleura, in the walls

of the heart, in the intestinal parietes, and beneath the peritoneum. There may be ecchymosis between the serous and muscular coats of the intestines. The mucous membrane of the intestines is reddened, swollen, and relaxed. Bloody serum may form in the serous cavities. The blood is of low specific gravity; there is a diminution in the red blood-corpuscles and an excess of fibrin. The joints are greatly enlarged. The gums will exhibit typical changes.

ETIOLOGY.

From insufficiency of or a total absence of fresh vegetables; from a decrease in the alkaline salts of the blood (salts of potash); from the constant use of salt meat, and long-continued exposure to privation, fatigue, and mental anxiety.

SYMPTOMS.

Anæmia—rapid and progressive.
Skin—has a dirty-looking, pallid, or earthy aspect.
General debility and cachexia.
Pains in the back and limbs.
Mental apathy and depression.
Tongue—large, flabby, and indented.
Constipation.
Spots—(petechial) first on the legs, and then extending over the surface.
Puffy swellings—in different parts of the body, due to deep-seated hemorrhages, especially in the popliteal space and elbows.
Bleeding and swelling of the gums, also ulceration or sloughing.
Loosening and dropping out of the teeth.
Fœtid odor of the breath.
Later on.
Puffiness of the face.
Anasarca of the extremities.
Dyspnœa.
Palpitation of the heart
Sudden syncope.
Offensive diarrhœa.
Disturbed vision.

Tinnitus aurium.
Vertigo.
Want of sleep.
Delirium.

CAUSES OF DEATH.

Death may occur from exhaustion or sudden syncope.

DIFFERENTIAL DIAGNOSIS.

From purpura.

PROGNOSIS.

Is good. These patients usually recover rapidly when fed on lemon-juice, fruits, and vegetable food.

TREATMENT.

Patient should have plenty of vegetables, lime-juice, and wine. Administer *tinct. ferri chlor.* in moderate doses. For the gums, a wash of *tannic acid* or *tincture of myrrh* in dilute glycerin; or alum, brandy, and water. Rub the body, or parts where the eruption is, with salt and whiskey.

PURPURA.

SYNONYMS.

Purpura hæmorrhagica; morbus maculosus Werlhofii.

DEFINITION.

Is an affection characterized by the extravasation of blood in the form of spots beneath the surface of the skin. These are unaffected by pressure. When they are small and round in shape they are termed "petechia;" when elongated in strips (like a whip), "vibices;" when diffused or irregular (like a bruise), "ecchymomata."

MORBID ANATOMY.

Is obscure. Hemorrhages, like those of the skin, are sometimes discovered in the sub-serous and sub-mucous tissues, and less so in the various organs. The primary morbid condition is probably in the capillary and other small vessels, rather than in the blood,

and the blood escapes into the tissues in consequence of capillary rupture.

ETIOLOGY.

It occurs at all ages, but chiefly in young children, who have delicate skins. It is thought by some to arise from bad hygienic surroundings, and in those who are under-fed, and sickly, and yet is observed among those who live with every comfort and good hygenic surroundings. One feature in this disease is that it is very apt to recur.

SYMPTOMS.

Usually ushered in with general malaise and fever.
Pain—in the lower extremities (especially in the legs).
Swelling of the affected limbs.
Skin—studded with red spots, usually about the size of a millet seed, which do not fade on pressure, but generally disappear in a few days.
Epistaxis—may occur.
Bleeding of the gums.
Loss of muscular strength.
Mental dejection.
In a severe case.
Bleeding from various organs.
Countenance—pallid.
Pulse—increased and jerking.
Tinnitus aurium.
Pupils—dilated.
Headache.
Muscæ volitantes.
Delirium.
Mania.
Convulsions or death.

CAUSES OF DEATH

Death may occur from asthenia or syncope.

TREATMENT.

In mild cases the patient usually gets well without any treat-

ment. Among the remedies which have been extensively used as styptics are *perchloride of iron*, *acetate of lead*, *arsenic*, *digitalis*, *turpentine*, and *gallic* and *sulphuric acids*. The body should be sponged with alum and brandy, or whiskey and water, at a temperature that is not chilling, and yet is sedative to the circulation.

GOUT.

SYNONYMS.

"Podagra;" and "arthritis."

MORBID ANATOMY.

There is an inflammation of the joints with deformity, and incrustation of the serous sacs of the joints, with a chalky material (urates of soda). It usually commences first in the joint of the great toe, although any joint may be affected. Fibroid degeneration of the blood-vessels may also ensue. The left ventricle of the heart may become hypertrophied.

ETIOLOGY.

It is said to be caused by an excess of uric acid in the system. It seems to arise from an hereditary tendency; from too high living; from the consumption of too much nitrogenized food and an insufficiency of oxygen introduced into the system; from plugging of the uriniferous tubules, producing subsequent atrophy of the kidneys. It usually occurs in men, after thirty years of age, who have led a luxurious and idle life.

SYMPTOMS.

Malaise.
Pain—severe, piercing, burning most often in the great toe. The attacks usually begin about midnight.
Joint—swollen and red.
Fever.
Pulse—full and bounding.
Skin—dry.

Thirst—intense.
Mental excitement.
Leg—swollen (by œdema).
Chalky deposits about the joints (occur late).
Hands and arms—become involved (later on).
Cachectic look.
If in the stomach.
Cardialgia.
Vomiting.
Hæmatemesis.
If in the brain.
Apoplexy, or circumscribed headache.
Dizziness.
If in the heart.
Irregular and feeble action.
Disturbed circulation.
Dyspnœa.
Fainting.

The bladder and kidney may be affected by the gouty diathesis.

CAUSES OF DEATH.

Death usually results from complications or internal gout

PROGNOSIS.

Is unfavorable as to a permanent cure. Patients are apt to die between the ages of forty-five and sixty years.

TREATMENT.

The patient should be placed under a certain regime. The following system will greatly aid in relieving the sufferer:

(1) The diet should consist principally of vegetables, soups, cream, and milk; meat may be taken *sparingly* once a day.

(2) Wines, spirits, and fermented liquors should be forbidden, and even tea and coffee.

(3) The patient should drink water freely (as it irrigates the kidneys), also Vichy, or any mineral water, and should eat lemons or take iced acid drinks.

Tincture of colchicum, gtt. xx.–xxx., may be given three times

a day or *colchicum* and *iodide of potassium* in combination; or *Reynold's* or *Seville's specific* or *Lee's lithontriptic pills;* these latter have proved to be a great source of comfort to the patient; or ℞ ipecac, hydrarg. chlor. mite, āā gr. i.; aloes, extractum colch. acet. ext. nucis vom., āā gr. ¼. Fiat pil. Sig. Give one every four hours till purged.

To relieve the pain, *opii gr. j.* or *morph. sulph.* gr. ¼ may be given.

DIPHTHERIA.

SYNONYMS.

"Pseudo-membranous angina;" "putrid sore throat;" "diphtheritis."

DEFINITION.

Is a disease characterized by a membranous exudation, which usually appears first on the tonsils. It may extend backward and upward, involving the posterior nares; or downward, involving the œsophagus and stomach, or respiratory tract. It may occur wherever mucous membranes exist, or even on a blistered surface.

MORBID ANATOMY.

On examination, the first thing that will be noticed is a reddening of the mucous membrane, which soon becomes purple in color (from passive congestion), and a slight infiltration of serum into the tissues (œdema) simultaneously appears. The epithelium covering the mucous membrane soon becomes cloudy and granular (resembling in appearance a light gauze which has been thrown over the mucous membrane). In the mildest cases the disease may stop at this stage, because this granular surface is of a low vital power, and cannot, therefore, organize.

In the next stage, the sub-epithelial layer becomes infiltrated with the same granular substance, which continues to progress until the whole of the mucous and sub-mucous tissues are involved.

The exudation varies in color and extent; it may, in one case, be pearly white, and cover over the surface; in another, it may

assume the buckskin appearance, covering the pharynx or tonsils; while in another it may be dark-gray in color, resembling gangrene, indicating extensive blood changes (the amount of exudation foretelling the severity of the disease). When the exudation is granular in character, the cells are changed tissue-cells, giving it the appearance of a mixed exudation. This is the lowest grade of inflammation, from which recovery is hardly possible; it resembles a caseous material, which cannot be re-absorbed, and is only removed by the removal of the epithelial covering. Should the exudation occur in the mucous and sub-epithelial tissues, and not be very abundant, it may be arrested by overcoming the poison. When this has been accomplished, simple epithelial and sub-epithelial cell development occurs, with a rapid suppurative process, terminating in a suppurative infiltration.

In addition to these local changes, on *post-mortem examination*, the blood will be observed to be much darker than normal. It undergoes rapid ammoniacal changes, and does not coagulate.

The heart will be found to be soft, and a granular degeneration of its muscular fibres will usually exist.

In the stomach, the mucous membrane will be found soft and œdematous.

In the intestines, the glands and Peyer's patches will be soft and enlarged.

The lungs will be congested (splenization), and the bronchi may contain the exudation.

The liver will also be congested, and the *spleen* enlarged and softened.

In addition to the foregoing changes in the blood and organs, there may also be those which resemble typhoid fever so closely that it will be almost impossible to distinguish the two diseases. Under these circumstances, the exudation must be present to decide it.

The first action of this disease is to destroy life, which it does by attacking the epithelial, sub-epithelial, and mucous tissues.

ETIOLOGY.

The questions which naturally arise are:

First.—Is diphtheria a contagious disease? There is no doubt that it is a contagious disease, and can be developed by inoculation in the same manner as small-pox.

Second.—Is it a constitutional disease primarily, and local secondarily, or vice versa? It cannot be well denied that it is first a constitutional, and, secondly, a local disease; for it seems proven there is a local manifestation of a constitutional development by the fact, that the disease cannot be destroyed by taking off the various layers of the mucous membrane. It is unquestionably a constitutional disease, and the local changes depend entirely upon the amount of infection.

Third.—Is it of spontaneous origin? It is like typhus and typhoid fevers, requiring a poison through decomposing animal matter, which fills the air. That it also occurs in connection with sewer poisoning there is no doubt, still the profession is very undecided on this point. It is a contagious disease, and has no apparent governing law. It may be communicated and produced, or transmitted, by the exhalations. It is claimed by some that this disease is due to some special germ. (This is not yet proven.)

SYMPTOMS.

Local.

Chills, fever, and active symptoms, with rapid pulse, in some cases precede the disease; while in other cases it comes on insidiously, and, only for the exudation, it might escape observation.

Exudation (*is characteristic*)—usually commences on the tonsils. It may extend backward and upward, or to the soft palate and pharynx, and into the posterior nares, or downward into the œsophagus and stomach, or into the larynx, and even to the lungs.

Pricking sensation in the tonsils, often extending down the throat.

Pain—in the throat and pricking sensation in the larynx.

Difficulty of deglutition—(from extension of the exudation into the œsophagus).

Voice—becomes changed, husky, and, finally, lost (from extension of exudation into the larynx).

Respiration—difficult both on inspiration and expiration, and wheezing in character (from extension of the exudation into the larynx).

Cough—abortive and stridulous (from extension of the exudation into the larynx).

Restlessness and jactitations—(indicating changes in the oxygenation in the blood).

Cyanosis.

Stupor and coma—(from excess of carbonic acid in the lungs).

Glands of the neck—enlarged (sub-maxillary gland is involved only when the exudation is on the tonsils, pharynx, and posterior nares, from extension of the poison through the lymphatic channels).

Puffing of the face may occur.

Death—may result from croupous laryngitis.

Constitutional—vary with the amount of poison in the system.

Pain in the bones.

Chilliness or violent chills.

Temperature—may rise to 105°–106°, or may not rise over 102°–103°.

Pulse—accelerated from 100 to 150. In some cases becomes irregular and intermittent.

Countenance—has a lethargic expression (in some cases).

Special symptoms—indicative of danger to the patient.

1. Extension of the exudation into the larynx—(from which few ever recover), producing change in the voice and a stridulous cough, which tells of danger and almost certain death.
2. Appearance of the exudation—which may be dark-gray, greenish, or black, involving the whole of the posterior walls of the pharynx and nares. It often emits an odor, resembling gangrene when the danger is great. Septic symptoms then develop, with fever and a high range of temperature, etc. Septicæmia may be ushered in by a chill, and followed by lethargy, stupor, peculiar offensive odor, disposition to a typhoid condition, and delirium

terminating in death (which is due to long-continued blood-poisoning).

3. Vomiting.—If it occur early, is not of very great importance; but, if late, then it is a grave symptom, indicating danger. It is not due to the shock of the poison, but to a secondary poison, and the patient often passes rapidly into collapse.
4. Nausea and diarrhœa—occurring late in the disease, are grave symptoms.
5. Pulse.—This is of three varieties:

   1st.—It may be rapid from the beginning and continue throughout at 140 to 180. This is due to the action of the poison on the pneumogastric nerve.

   2d.—It may be rapid from the beginning and suddenly go down to 60 or 80, and afterward become intermittent. This is due to the action of the poison on the sympathetic system.

   3d.—There may be marked irregularity from the onset. It may be intermittent and rapid at first, then becoming slow, or vice versa. This is due to the action of the poison on the cerebro-spinal system.
6. Paralysis of the muscles of the larynx, neck, arm, etc.
7. Low temperature and cold surface, accompanied by a cyanotic condition. Even if the pulse be rapid and not intermittent and irregular, this is unfavorable. It denotes action of the poison on the nerve-centres.

SEQUELÆ.

The most frequent is that of paralysis of the pharynx, larynx, arms, lower extremities, bladder, penis, etc. There are no medical means which can restore the normal power, the action of the muscles seems to be lost for a certain time, and then to return to their normal condition; thus clearly proving that there is no great structural lesion in the nerve-centres.

Another sequela is parenchymatous nephritis, which commences in the uriniferous tubules. These are filled with an exudation the same as in other infectious diseases. There may be

a granular exudation of the uriniferous tubules, going on from one step to another until it produces total destruction and intense engorgement of the kidneys, resulting in blood in the urine, etc. Uræmic symptoms will then be developed, and the patient may die in convulsions from uræmic poisoning. The diphtheritic renal changes are sometimes the cause of chronic Bright's, having its origin in a parenchymatous inflammation from the result of diphtheritic inflammatory changes.

COMPLICATIONS.

Endocarditis.—This is usually of an *ulcerative type*, and occurs after the exudation has disappeared. It requires more than a simple irritation of the blood passing over the endo-cardial surface to produce it, and denotes very extensive constitutional poisoning.

Meningitis.—If it occur at all, appears early in the disease.

Hemorrhage of the bowels.

Jaundice.—(From extension of the exudation along the line of the hepatic duct). The inflammation occurs in the smallest tubules of the liver.

The inflammatory process may occur in the smallest bronchi in the lungs, extending downward from the larynx, or it may commence in the bronchi and extend upward. There is no mucous membrane in the body that may not become involved.

DIFFERENTIAL DIAGNOSIS.

Rests entirely upon the presence of an exudation, which usually shows itself first on the pharynx. Unless the exudation be perceived on some mucous surface, and there be marked constitutional disturbances, the diagnosis of diphtheria cannot be made. It may be diagnosed from catarrhal pharyngitis, membranous croup, typhus or typhoid fevers, and ulcerative endo-carditis.

In catarrhal pharyngitis—you have no gray exudation, and the patient gets well in from four to five days.

In membranous croup—the membranes are superficial, and there are no constitutional disturbances. They are simply local.

In typhus and typhoid fevers.—It is very difficult to decide such cases as these, for, whenever the exudations occur, it will always be along the intestinal tract, and all that can be done,

under these circumstances, is to watch the discharges for two or three days, and also the matter that may be vomited, when the exudations may be perceived.

In ulcerative endocarditis.—The patient first complains of sore-throat; he is seized with pains in the back, perhaps hemiplegia, and suddenly becomes unconscious; the pulse will be irregular, a mumbling delirium may be present. On examining the heart it will be found to have a tumbling motion and extensive valvular lesion; under these circumstances examine the posterior nares, when you will be certain to find a small mucous patch of exudation, which will confirm your diagnosis.

PROGNOSIS.

Depends upon whether there is an epidemic raging, and also upon the stage of the epidemic; the fatality being worse in the early stage. It is apt to demolish a whole township of children. *Age* is an important element of prognosis; for, the younger the the child, the worse the prognosis, as death seems to result from obstruction in the pharynx (and consequently from an inability to feed the child), and further, from the excess of poison in the constitution. When it attacks the adult, the prognosis is more favorable. The *character of the exudation* is an aid in the prognosis; for, the darker, and thicker, and more extensive the exudation, the worse the prognosis. If it attack the posterior nares, and extend upward, if the glands become enlarged, and the constitutional symptoms are severe, the prognosis is grave; but, so long as the exudation confines itself to the fauces and anterior pillars of the pharynx, and spreads, *but is not thick*, even though other symptoms be severe, the prognosis is favorable. Should the exudation extend downward into the larynx and respiratory tract; or into the œsophagus, it will then be unfavorable.

If *the exhalations* from the throat be offensive, and, following the shock, there be evidences of septic poisoning, the prognosis is bad. Should *pharyngeal paralysis* occur before the exudation has disappeared, recovery is hardly possible, as deglutition becomes involved, and death resulting from inanition. The paralysis which comes on after the disappearance of the exudation is

usually recovered from, yet there is one form of paralysis which destroys life, and that is *when the intercostal muscles are paralyzed*, accompanying paralysis of the diaphragm (since the patient then dies from inability to respire).

Should the slow pulse come on after a fast pulse, it may be recovered from; but, if in addition to the *slow pulse* it becomes *intermittent*, the prognosis is bad.

TREATMENT.

May be divided under four headings:

1. *Hygienic.*
2. *External local.*
3. *Internal local.*
4. *Constitutional.*

Hygienic.—The patient should be placed in a large, well-ventilated room, the temperature of which should range from 68° to 72°. Great care should be exercised in regard to cleanliness, for there is no disease in which cleanliness is so important. The bed and body-linen should be carefully cleansed; the patient should be quarantined, and all other children removed from the house, or kept away from the apartment. It is best to place the patient in an upper sunny room, provided, if possible, with an open fireplace; everything which can be dispensed with, such as books, clothing, carpets, window curtains, etc., should be removed, because when once the patient has entered the room, nothing can with safety be removed until *disinfected or fumigated.* One or two adults should take the entire charge of the patient, under no circumstances coming in contact with other persons, especially children. Great care should be observed *not to inhale the breath of the patient*, and parents should be prevented from kissing the child. All soiled linen, and also every article used in the room, should be thoroughly disinfected before it leaves: this can be easily accomplished by obtaining one pound of *sulphate of zinc:* place eight tablespoonfuls of this, with four of common salt, in an ordinary water pail, and, to them add one gallon of boiling water. This disinfectant solution must be kept in the sick-room, and into it should be placed, and kept for one hour, every article

of soiled clothing, bedding, etc.; when they are removed from this they must be put into boiling water before being washed. The dishes and spoons used by the patient should be put into boiling water before they are permitted to leave the room. It must be borne in mind that every article which is in the room can convey the disease, and that nothing should go from it until the poison, which they might carry, has been destroyed.

Be very careful when making an examination that the breath is not inhaled, nor the sputa gain entrance into your mouth. *Avoid, as much as possible, manipulation and inspection of the throat*, as it produces irritation, and will necessarily increase the exudation ; examination should always be made with the *utmost care, and not too frequently.* One of the principal features in the treatment of this disease is *absolute rest* in the *recumbent* position, and the patient should not be allowed to move about or walk. The diet should consist simply of *milk*, and given in small quantities. The body should be carefully sponged, and the nasal passages syringed out once a day, with a half per cent solution of carbolic acid, but care should be taken not to exhaust the patient. The windows of the sleeping-room should be opened, as long as possible, every day, as *fresh air* is about the best disinfectant; but draughts and chilling the patient should be carefully avoided, and see that a fire is kept burning in the room, day and night, during cold weather.

External local.—This consists of

(1) *Blood-letting*—by the application of leeches to the angle of the jaw.
(2) *Cold*—by means of the ice-bag.
(3) *Counter irritation*—in the form of blisters, etc.
(4) *Hot applications and hot poultices.*

Of these, the preference must be given to the *hot application* (which should be covered over with oiled-silk when applied) and *hot poultices*, because, so long as there is a granular exudation into the membranes, and it can be converted into a serous, the sooner it will cease. These *hasten suppuration*, and should, therefore, be *applied regularly and carefully* at the seat of exudation.

Internal local.

1. *Mechanical.*
2. *Escharotics.*
3. *Astringent.*
4. Antiseptic.

With regard to the first three methods, it is hardly necessary to assert that they are only detrimental, as they tend to aggravate the part and cause the exudation to infiltrate the deeper tissues, and produce gangrene and sloughing. *The exudation should never be removed,* unless you find a part that has sloughed and is pendant; it should then be drawn forward and cut off. The question naturally arises; you require to start a suppurative process under the exudation. What is to be done? What shall be used? *Steam* should be used, as it is the most grateful course that can be pursued for the patient, because it tends to produce suppuration; therefore, apply *hot poultices externally,* and cause *steam to be inhaled;* and, so soon as the removal of the exudation is observed, you know your patient has had a local affection.

What is to be done with the septic poison? Use *antiseptics* by *means of the spray,* with a weak solution of *carbolic acid* or *chlorine water,* and a very weak solution of the *muriate tincture of iron;* or give the patient muriate tincture of iron, gtt. v., with glycerin; or chlorate of potash, gr. v., with glycerin, and let him swallow it, as it acts in the same way as the spray when that plan cannot be resorted to.

Constitutional.—From the onset there are evidences of failure of vital power and loss of all symptoms of vitality, the patient dying, as they do in all infectious diseases, from the direct effect of the over-charging of the poison. The disease must, therefore, be treated in all ways, as the other infectious diseases. *Muriate tincture of iron* should be given from the commencement. As stimulants, *alcohol* or *brandy* stand first, but should not be used unless the system is very much disturbed constitutionally, as they are not required; but, so soon as you find the patient becoming apathetic, the pulse feeble, and more frequent, and perhaps becoming irregular, the tongue dry, and the exudation dark and offensive, then crowd your stimulants, the first few doses of which should be

carefully watched, and it may even be necessary to give as much as a tablespoonful every hour to a young child. *Milk* should be the diet upon which the patient should be fed; if it cannot be swallowed, it must be given by the rectum. Should there be restlessness and jactitations, *opium* may be given in moderate doses, but not sufficient to produce narcotism.

In the laryngeal or pharyngeal complications *malt liquors* should be given and as much as the patient will take, all other methods of treatment being of no avail.

In cardiac diseases *opium judiciously administered* will have a beneficial effect, but in children the *only* preparations which should be given are *Pulv. Doveri.* and *tinct. opii camph.*

When kidney complications occur they must be treated in the same manner as acute parenchymatous nephritis.

RELAPSING FEVER.

SYNONYM.

Famine fever.

DEFINITION.

Is an epidemic fever, characterised by a sudden invasion and persistent high febrile symptoms without remisssion; temperature rising to 107°-108°. There is a rapid subsidence within a week and then a relapse, usually, within seven days after the first attack. The temperature in this fever rises higher than in typhoid. It is claimed that the disease is due to spirillœ within the blood.

MORBID ANATOMY.

The lesions occurring in this disease will be found in the spleen, liver, kidneys, intestines, mucous membranes and the blood.

Spleen—may be increased in size; its capsule thickened, smooth tense and slightly cloudy; its trabeculœ increased in size; and the Malpighian tufts more prominent than normal, or it may be enlarged, soft and flabby.

Liver—may be enlarged; the structural changes will be the same as those in the spleen. The gall-bladder may be distended with bile of a dark yellow color.

Kidneys—increased in size which is due to congestion of the cortical substance and a granular infiltration of the epithelium of the uriniferous tubules.

Intestines—glandular follicles will be enlarged. The solitary glands are principally affected and Peyer's patches may have the shaven-beard appearance. The mesenteric glands may resemble those of typhus or typhoid fever.

Mucous membranes.—Those of the stomach and intestines are principally affected having small spots of blood-extravasation upon their surfaces, but these extravasations may also occur on the mucous membranes of the bronchial tubes.

Blood—coagulates imperfectly. Some authorities claim that spirillæ are always present in the blood of patients afflicted with this fever.

ETIOLOGY.

It is said to oe due to spirillæ in the blood having the power of developing the fever. It seems to be developed by squalor, poor quality of food, and bad hygienic surroundings. It may be traced to direct contagion.

SYMPTOMS.

Advent is sudden.

Chill—distinct.

Frontal headache (which increases with the fever).

Pain in the muscles and joints—(which increases with the fever).

Nausea.

Vomiting—at first, contents of the stomach, then a yellowish material, and subsequently of a dark color resembling the black vomit of yellow fever.

Temperature—rises rapidly especially during the first twenty-four hours; may reach 104° or even 109° and may last for two or three days.

Pulse—may reach 140 to 160 during the first twenty-four hours and is small and compressible.

Respiration—hurried and irregular.

Great prostration and *emaciation.*

Weight and uneasiness in the abdomen, especially in the right hypochondrium.

Liver—enlarged and tender.
Spleen—enlarged and tender.
Muscular pains—Severe and increased by motion and pressure and is of a piercing and lancinating character, and patient lies perfectly quiet and sleepless in consequence.
Delirium—may occur and be active in form.
Pupils—may be irregular.
Photophobia—may occur.
Jaundice—may occur with vomiting and diarrhœa.
Eruption—may occur, resembling roseola.
Perspiration—profuse about the seventh or eighth day of the fever when the temperature falls and the symptoms abate.
Hemorrhage—from the nose, bowels, or intestines may occur.
Relapse—may occur from the third to the seventh day after the symptoms abate which last about four days.
Œdema of the feet—may occur during convalescence, due to great anæmia.

CAUSES OF DEATH.

Deaths usually occur from complications and not from the disease as from diarrhœa and dysentery during convalescence; from pachymeningitis; from hemorrhage from some of the mucous surfaces; from *syncope* during relapse; from bronchitis, pneumonia or pulmonary complication; from suppression of urine producing uræmia.

COMPLICATIONS.

Pneumonia which is often double; sudden collapse; post febrile ophthalmia attacking most frequently the right eye; diarrhœa and dysentery.

DIFFERENTIAL DIAGNOSIS.

Is not difficult. It may be confounded with typhus fever; typhoid fever; remittent fever; yellow fever; dengue fever; small pox (before the eruption); and measles. It differs from them all in the suddenness of its invasion; short duration of its primary fever; high temperature and pulse; termination in a crisis; the occurrence of a relapse between the third and fifth days and constant muscular and arthritic pains.

PROGNOSIS.

Is always good unless complications exist, the rate of mortality being lower than in measles.

TREATMENT.

This disease has been experimented upon by almost every drug in order to control the fever ; the decision now arrived at is that these patients progress more favorably when placed under *the best hygienic surroundings* with free ventilation, a mild diet and *no medication* whatever. During the primary fever the patient should be kept in bed and great care should be taken to guard against syncope, Should heart failure occur *digitalis* and *stimulants* must be administered.

Test to discover Spirillœ in the blood (by Albrechs, of St. Petersburg).

1st. Dry a drop of blood on a slide.

2d. Put a drop of acetic acid on the slide which eats away the fibrin and blood-corpuscles.

3d. Wash off the slide with care so as to get rid of the dissolved fibrin and blood-corpuscles.

4th. Dry the specimen and examine under the microscope, when the spirillæ will be observed, if present

DISEASES OF THE URINARY ORGANS.

RENAL CONGESTION.

SYNONYM.

Hyperæmia of the kidney.

MORBID ANATOMY.

In active hyperæmia, the kidneys may be found very much increased in size, in both the cortical or medullary portion, and this increase will be most marked at the base of the pyramids. The kidneys will be unnaturally dark in color. The capsules will be non-adherent, the surfaces smooth, and the organs softer and more moist than normal.

On section, dark spots will be seen corresponding to the Malpighian tufts. A fluid will exude on section of the organ (half blood and half serum), due to œdema associated with congestion of the vessels. The congestion occurs in the arteries and Malpighian tufts before the veins are involved. The changes in the organ are confined to engorgement of the blood-vessels, and an infiltration of serum into the intertubular structure in the cortical portion.

In *passive* or *mechanical hyperæmia* (especially when occurring in chronic cardiac disease), the kidneys are not much increased in size, but are of a stony-hard character and smooth. The capsules are non-adherent; their surface is smooth; and they are of an uniform red color.

On section, the medullary is darker than the cortical portion. The Malpighian tufts are not prominent, but are simply filled with blood. The veins are dilated and hard; the hardness is due to a constant dilated condition of the efferent capillaries. There are not, necessarily, inflammatory changes in this form of kidney, but they may be accompanied by inflammation of the uriniferous tubes. There may be more or less change in the tubules, characteristic of simple catarrh of their lining structures.

ETIOLOGY.

Active congestion is produced by exposure to sudden changes;

to blood-poisons, such as scarlatina, diphtheria, typhus fever, malaria; and to irritation of the urinary passages by cantharides, turpentine, nitre, copaiba. It may result also from diabetes, cholæmia, morbid formations in the organ, traumatism, disease of the pelvis of the kidney, and cardiac hypertrophy (on account of an accelerated, or too forcible heart's action).

Passive congestion is produced by diseases of the heart or lungs; mechanical obstruction in the thorax or abdomen; venous obstruction to the right auricle, such as pressure on the vena cava above the renal vein, or on the renal vein; arterial obstruction from the left ventricle; valvular lesions (especially mitral regurgitation, or tricuspid insufficiency); structural diseases of the cardiac walls; obstruction to the pulmonary circulation, as occurs in emphysema, pleuritic effusions, pneumo-thorax, fluid in the pleural cavity; pressure on the inferior vena cava, as occurs in pregnancy and abdominal tumors; mediastinal tumors pressing on the pulmonary artery or vein; weak heart, from exhaustion and old age; surgical operations on the urethra (from nervous causes); calculi in the kidney; extension of inflammation from the urethra and bladder; and pressure on the heart from pericardial effusions.

SYMPTOMS.

Decrease in the quantity of urine.
Increase in the specific gravity.
Albumen and traces of blood. (If albumen alone, it usually indicates inflammation.)
Blood-casts of the uriniferous tubes (rare).

PROGNOSIS.

Is bad, if it occur in connection with cardiac disease, and also in the congestive malarial fever of hot climates. Renal congestion may be followed by inflammation of the uriniferous tubes and development of Bright's disease.

TREATMENT.

For active hyperæmia.—The patient must be kept in bed, with the temperature of the room about 75°. A dozen dry or wet cups should be applied over the lumbar region. *Diluent drinks* may

be freely indulged in. Administer one or two drastic purgatives, and *avoid* stimulants. Induce moderate diaphoresis.

For passive hyperæmia.—Mild counter-irritants, such as three or four cups, and *no blisters*, may be used. A *brisk cathartic* may occasionally be given, or a dose of *calomel*, in combination with some other purgative. Should there be great renal disturbance, *digitalis* may be given freely. If in connection with pregnancy, cupping will be found serviceable.

RENAL HEMORRHAGE.

MORBID ANATOMY.

The changes are much the same as those of renal congestion. The hemorrhage is most frequently due to embolism and infarction, traumatism, new-growths, or renal calculi. Congestion always takes places in the vessels which surround an infarction. Little masses, like ecchymotic spots, may often be observed in the cortical substance; and these are due to an effusion of blood into the uriniferous tubules, or interstitial tissue. *On section*, blood often flows freely from the uriniferous tubules. The seat of the hemorrhage is usually marked by the development of hard uniform masses in the cortical portion; these infarctions are wedge-shaped, with their apex toward the hilum of the kidney, and their base toward the surface; they may be capillary, and very small in size. At first they are dark-red, gradually becoming lighter in color. In the centre they may have a cheesy appearance. Changes often take place in the uriniferous tubes in the region of these capillaries, thus causing congestion and the production of lymphoid cells, which may undergo rapid purulent transformation, producing abscesses. The infarctions may increase in size, and become necrotic, producing gangrene. Capillary thrombi are sometimes formed, and may become numerous; this is due to the slowness of the capillary circulation.

ETIOLOGY.

Intense active congestion, resulting in an engorgement of the kidneys with blood, and rupture of the capillaries (occurring in

scarlet, typhus, and malaria fevers; injuries, wounds, or contusions of the kidney; cancerous development: renal calculi in the pelvis of the kidney; purpura; scurvy; cardiac diseases (producing passive obstructive congestion); infarction (due to renal embolism or capillary thrombosis).

SYMPTOMS.

Urine.—This is the only means of ascertaining a renal hemorrhage, as the blood will be effused into the uriniferous tubules or hilum of the kidney, and discharged into the bladder.

If it occur from renal calculi—hemorrhage is liable to take place at every violent exertion.

If it occur from cancer or tumor—it is usually profuse in quantity and persistent.

If it occur from infectious diseases—it is slight, and perhaps only recognized by the microscope.

If it occur from malaria—it is profuse and periodic.

If it occur from infarction—there are chills (as it is usually due to pyæmia).

PROGNOSIS.

If it occur from renal calculi or cancer, the prognosis will be bad; if from infectious diseases, it is merely an indication of intense hyperæmia; and, if from infarction, it is always attended with danger.

TREATMENT.

The patient should be kept *absolutely* at rest when hemorrhage occurs. *Ice-bags* should be applied to the lumbar region, and *styptics*, such as tannic acid, be given *internally*, as it is expelled through the kidneys in the form of gallic acid, if the hemorrhage be profuse, and there be danger of exhaustion. In some cases *ergot* may be given in large doses. If it be due to malarial poisoning, large doses of *quinine* are indicated.

URÆMIA.

In this disease, the symptoms are divided into two classes.

1st. *Urœmic convulsions*—consisting of twitching and epilepti-

form convulsions of the voluntary muscles, ushered in by headache, nausea, and vomiting.

2d. *Urœmic coma*—which is a state of insensibility, ushered in by headache, drowsiness, or convulsions.

Both of these conditions are due to failure of the kidney to eliminate urea, and hence accumulation of poisonous matter in the blood. This may occur in almost any disease.

ETIOLOGY.

It may be due to a complete or partial arrest of the urinary secretions, or to the retention of the united products of all the nitrogenized effete matter, usually eliminated by the kidneys; or to urea, which, when in excess, is an irritant poison, acting primarily on the cerebro-spinal centres, and through them interfering more or less with the functions of organic life.

SYMPTOMS.

In acute urœmia.

Œdema—in various parts of the body.
Restlessness.
Impaired vision.
Headache.
Nausea.
Vomiting.
Drowsiness.
Countenance—pale, waxy, dingy appearance.
Urine—scanty, high-colored, and bloody; it contains albumen and casts.
Violent convulsions—from excess of urea in the circulation, when the
    Face—becomes livid.
    Eyes—glassy.
    Pupils—contracted or dilated.
    Frothy or bloody mucus around the mouth.
    Perspiration—is of an urinous odor.
    Temperature—elevated (it may reach 107°).
    Pulse—accelerated.
Coma—comes on gradually; resembles an attack of cerebral

apoplexy; it may be extremely profound, or the patient may be easily aroused, or even return suddenly to consciousness. Attempts to speak and swallow fluids are performed with difficulty.

Respiration—is slow labored, and stertorous during the coma.

Pupils—regularly dilated.

Pulse—rapid and weak.

Temperature—rises, and gradually falls below normal.

In chronic uræmia.

Headache—a prominent symptom.

Insomnia.

Lassitude.

Countenance—peculiar pallor, waxy, etc.

Skin—dry or moist, and has an urinous odor.

Muscæ volitantes—due often to œdema of the retina.

Nausea.

Vomiting—sometimes.

Restlessness.

Impaired digestion.

Œdema—first about the eyes, then the ankles, and extending upward.

Œdema of the lungs.

General anasarca.

Changes in the urine—which depend upon the type of kidney disease present.

Convulsions.

Serous inflammation—especially endocarditis and meningitis in the later stages.

DIFFERENTIAL DIAGNOSIS.

From epileptic seizure; cerebral apoplexy; ammonæmia; hysterical convulsions; opium poisoning; and cholæmic convulsions.

Epileptic seizure.

One side convulsed more than the other.

Loss of consciousness. Reflex sensibility and no rise in temperature.

Deep sleep follows the seizure.

Urœmic convulsions.
Both sides equally convulsed.
No loss of consciousness.
Rise in temperature.
Deep coma follows the seizure.

Cerebral apoplexy.
Coma precedes the convulsions.
Paralysis (hemiplegia) can usually be detected.
Clonic spasms of the affected side.
Urinary symptoms of uræmia absent.

Urœmic convulsions.
Convulsions precede the coma.
No paralysis.
Spasms of both sides equally.
Urine will decide the case.

Ammonœmia.
Urine, breath, and perspiration are all ammoniacal.
Mucous membrane of the mouth is dry and shining.
Sallow complexion.
Frequent chills.
No dropsy.
Vomiting—rare.
Convulsions—rare.
Coma precedes death.

Urœmia.
Perspiration—has an urinous odor.
Countenance—pale, and of a waxy appearance.
Chills—possible.
Dropsy.
Vomiting.
Convulsions.

Hysterical convulsions.
Patient falls (often with a scream) into a convulsive tetanic, or cataleptic condition.
Pupils—normal and constant blinking of eyelids.
Temperature—normal.
Pulse—normal.

Limbs—jerked irregularly.
No lividity of the countenance.
Breathing—spasmodic, and a choking sensation is often complained of.
Discharge of large quantity of pale urine after the attack.

Uræmic convulsions.

Convulsions are preceded by headache, etc.
Pupils—regularly dilated, or contracted.
Temperature—accelerated.
Pulse—accelerated.
Jerking is regular.
Countenance—livid.
Respiration—labored, slow, and stertorous.
Urine—will decide the case.

Opium poisoning.

Temperature—usually below normal.
Respiration—slow and regular.
Pupils—uniformly contracted (pin-point pupils).

Uræmic coma.

Temperature—elevated 100°.
Respiration—slow, labored, and stertorous.
Pupils—uniformly dilated.

Cholæmic convulsions.

Produced from overcharge of bile in the blood.
The convulsions are preceded or accompanied by jaundice.

CAUSES OF DEATH.

Death results from the action of urea on, and overwhelming of, the nerve-centres, producing changes in the blood, which interfere and arrest oxygenation; there are then structural changes taking place in the tissues, rendering them incapable of supporting life.

PROGNOSIS.

Depends upon the amount of urea in the blood, and the time it has been accumulating.

TREATMENT.

The patient should have *absolute* rest and free elimination, procured either by the skin or bowels, or both. *Diaphoresis* may be accomplished with hot-air baths. Drastic purgatives, as *elaterium*, *scammony*, or *croton oil*, may be administered. *Digitalis* is the best diuretic that can be given; it strengthens the heart's action and the eliminating power of the kidneys, since the blood-pressure is increased. *Inf. of English leaves*, dose ʒ ss., may be taken *every three hours* for the first twenty-four hours, or until the specific effect of the drug is produced. For uræmic convulsions, *morphine* should be administered *hypodermically;* ♏ x. at first, and if that does not arrest them, give ♏ xx. every two hours, to control the convulsions. It arrests the muscular spasm by counter-acting the effect of the uræmic poison on the nerve-centres, and establishes diaphoresis, and it further facilitates the diuretic action of digitalis.

BRIGHT'S DISEASE.

The three distinct elements which are primarily or secondarily involved in the morbid changes of the kidney are (1) the uriniferous tubules; (2) the blood-vessels in the walls); and (3) the intertubular connective-tissue.

The various forms of Bright's disease may be classified under three heads (from a pathological basis), as follows:

1. Those which are *inflammatory* in their nature, and commence in the uriniferous tubules, designated as "*parenchymatous nephritis.*"

2. Those which are *non-inflammatory* in their nature, and commence in the walls of the blood-vessels, designated as the "*amyloid variety.*"

3. Where the changes commence in the intertubular tissue, designated as the "*cirrhotic type.*"

PARENCHYMATOUS NEPHRITIS.

This is the most common form, and is of an inflammatory nature. It may be of either short or long duration, and may,

therefore, be subdivided into the acute and chronic form. It has three stages.

STAGES.

1st Stage. *Active inflammation* — called "acute desquamative tubular nephritis," or "acute parenchymatous nephritis."

2d Stage. *Degeneration*—which may be either fatty or granular, and is called by some authors the "*fatty*" or "*large white kidney.*"

3d Stage. *Atrophy* of the organ.

MORBID ANATOMY.

First stage (inflammation).—This may be either catarrhal, croupous, or desquamative in type. The kidneys are increased in size; their capsules are non-adherent; their surfaces are smooth or mottled; they are usually of a dark and purplish color, dotted with spots of ecchymosis, and are firm on pressure.

On longitudinal section of the organ.—The cortical portion is seen to be increased in volume and dotted over with dark or red spots, which correspond to the congested Malpighian tufts. The cortical substance between the tufts may be paler than normal. There will usually be engorgement, well-marked at the base of the pyramids (at the junction of the cortical and medullary portion); the medullary portion will be darker than normal, with alternating red and white lines (the light lines denote changes in the uriniferous tubes). The lining membrane of the pelvis is somewhat congested. The well-marked engorgement at the base of the pyramids exists, on account of the venous arcades being situated over the base of each pyramid.

On microscopical examination, the tubules will be found to be the seat of a simple catarrhal process. The epithelial lining may be partially or completely lifted, and may have disappeared, while the tubules may be more or less filled with cells corresponding to new cell-formation (a simple catarrhal inflammation). The centre of the tubes may also contain a hyaline material (which is coagulated fibrin), or this hyaline material may be surrounded by epithelium and blood-globules. (It is an inflammatory exudation, resembling that of croupous inflammation elsewhere.) These

changes usually commence in the epithelial cells of the tubes; the cells become cloudy; their nuclei disappear; they become distended and granular; and, finally these changes are followed by desquamation. The tubes may thus be filled with broken-down epithelium and fatty matter (the latter condition indicating *chronic* desquamative nephritis). In the first stage of the inflammatory variety you may have one or all of these conditions present in the uriniferous tubules, and also new cell-formation in the in the intertubular tissue.

Second stage (fatty or granular kidney).—This is described by some authors as a special type of kidney. The kidneys are enlarged; the capsules non-adherent; the surfaces smooth; the organ paler in color and softer than normal (when fatty degeneration is going on), and presenting a yellow or mottled appearance. The enlargement is due to an increase in volume of the cortical substance (as shown on section of the kidney), which is of a pale yellowish-white color. Under the *microscope*, the organ shows more or less fat in the tubes, and a diminished vascularity of the kidney. The changes occur chiefly in the convoluted tubes of the cortical substance adjoining the Malpighian bodies. The tubules are distended with oil-globules, and large hyaline casts may be found in them. Atrophy, or granular degeneration of the kidney follows (if the circulation and nutrition of the organ be interfered with) since this stage is associated with, or the result of long-continued active or passive renal congestion.

Third stage.—After the transformation (fatty) of the epithelial elements in the second stage, cellular elements are developed in the walls of the tubes and intertubular tissue. Organization and contraction of this new connective-tissue occurs, compressing the blood-vessels. The tubules thicken, and atrophy of the kidney substance, together with fatty and granular degeneration of the contents of the tubules and the epithelium, is developed.

The *microscope* in this stage shows excessive development of the new connective-tissue and constriction of the tubules, causing development of cysts. The kidneys are uneven and nodular, and smaller and harder than normal; the capsule is thickened and adherent, and, when removed, tears off part of the kidney. The

cortical substance will be seen to be decreased, and sometimes to have almost disappeared; the tubes have collapsed, and cysts are found to have developed. The principal changes are thickening of the walls of the blood-vessels; more or less obliteration of the uriniferous tubules, and disappearance of the epithelial lining; evidences of a previous fatty and granular degeneration, and a marked increase in the walls of the tubules and the capsules of the Malpighian tufts.

In summary, the following changes may thus be said to take place in the several stages of this form of nephritis (the inflammatory form):

1. There may be more or less *intense congestion*, with very active proliferation and desquamation of the epithelium, which lines the tubules (causing the tubes to be blocked up and distended).

2. This excessive engorgement may produce a rupture of the capillary vessels and an escape of blood-globules into the tubes, and an effusion of fibrin from the interrupted circulation. This latter exudation coagulates in the tubes, and mixes with the epithelial cells and blood-globules (hyaline material).

3. The contents of the tubules (epithelial cells) become the seat of a fatty degenerative process. Their nuclei soon disappear, and the tubules become distended and filled with a mass consisting of broken-down epithelium matter and fat.

ETIOLOGY.

PREDISPOSING CAUSES.

From exposure to sudden changes; from alcohol, "going on a spree," and sitting in a draught; from irritation and inflammation of the uriniferous tubules, produced through excessive labor of the kidneys to throw off the accumulated excrementitious material (the power of elimination being arrested through the defective action of the skin); from inflammation due to reflex influence of the nervous system; from additional work and some peculiar shock through the sympathetic; from renal hyperæmia, and its various causes; from inflammation of other organs.

EXCITING CAUSES.

(1) Blood-poisoning (as evidenced in the renal hyperæmia following fevers, scarlatina, typhus, diphtheria, measles, pyæmia, rheumatism; and in the direct irritation of the tubuli uriniferi, following the use of copaiba, turpentine, cantharides, etc.); (2) mechanical interference with the circulation of the kidney, as occurs in pregnancy and cardiac diseases; (3) senile decay, pneumonia, extension of inflammation from adjacent structures, etc.

SYMPTOMS.

In the mild form, or simple catarrh of the uriniferous tubules.

Œdema of the face.

Œdema of the feet and ankles (occurs early if heart lesions exist).

Restlessness.

Headache—constant and increasing (due to irritating effects of the accumulated urea).

Urine—scanty, high-colored, and of high specific gravity.

Frequent micturition (if the bladder experiences irritation).

Nausea, Vomiting, } evidences of the poisonous effects of urea.

Perspiration—has an urinous odor (if the poisoning by urea be rapid).

Pain—in the back and loins.

Drowsiness.

Dry skin.

Pulse—accelerated and irritable—firm and hard.

Rise in temperature (denoting fever).

As a severe type develops from a mild form.

Œdema—all over the body.

Pulmonary congestion.

Expectoration—watery, streaked with blood.

Dyspnœa—(from pulmonary œdema, or uræmia affecting the respiratory centres).

Countenance—pale and waxy.

Water-logged condition (the dropsy accumulating gradually as the case goes on).

Œdema of the scrotum and penis—(from blood-poisoning).
Great restlessness—(from blood-poisoning of nerve-centres).
Muscular twitchings—(from blood-poison of motor centres).
Convulsions—from the blood-poison, creating irritation of the cortex, or from pressure of œdema.
Coma—from the blood-poison acting upon the nerve-centres to a marked extent.
Death—from the blood-poison, creating œdema of lungs, heart complications, meningeal inflammation, etc.

In the chronic variety.

If commencing with an acute type, the following symptoms may be developed:

General anasarca.
Pulmonary œdema.
Countenance—pale and waxy (like cancer).
Urine—passed in large quantities.
Appetite returns,
Nausea—disappears,
Restlessness—disappears,
Sleep becomes refreshing,
} as the symptoms of *acute uræmia* subside, and the disease assumes a less active form of blood-poisoning.
The patient is thus passing on to the third stage, and becomes a more or less confirmed invalid.
Œdema of the feet is always present (to a greater or less degree).
Cardiac hypertrophy develops (on account of changes in the capillary vessels, and from poor nutrition to the heart, which necessitates an increase of size to insure the normal power of that organ).

In very acute cases—uræmia may be ushered in by

Chills.
Temperature—markedly elevated, 106°.
Pain—well marked along the back and ureters.
Marked œdema of the face.
Urine—completely suppressed or greatly diminished in quantity, or even bloody.
Nervous system—disturbed to a marked degree.

Testicles—retracted (if the kidneys be excessively irritated, even if a calculus be not present).

Delirium.

Coma—will probably develop rapidly.

Death may result in two or three days.

The main cause to which most of these symptoms may be attributed *is urea*, in abnormal quantities in the blood (producing the phenomena which attend the development of the different forms).

The *principal symptoms* that always present themselves for consideration *are* those in connection with the *changes in the urine*, the development of *dropsy*, and the *nervous phenomena*. These may be thus hastily summarized:

URINE.

In the First Stage.

(*Stage of congestion and exudation in the uriniferous tubules*).

Scanty and high-colored.

High specific gravity (1.030).

Albumen (usually about one-half of the specimen examined).

Casts—epithelial, small hyaline, blood-casts, and blood-globules.

In the Second Stage.

(*Stage of exudation and fatty degeneration*).

More abundant.

Not so highly colored.

Lower specific gravity (1.005).

Albumen (usually about one-third of the specimen examined).

Casts—oil-globules and fatty, in addition to those in the first stage.

In the Third Stage.

(*Stage of degeneration and atrophy*).

Greatly increased, and becomes pale.

Specific gravity usually about 1.010.

Albumen—(very slight quantity, if any).

Casts—fine granular and large hyaline.

Dropsy.

Is caused by the excess of urea acting upon the capillaries and composition of blood. It can thus be explained·

(1) by hydræmia, and

(2) by imperfect nutrition of the heart (when there is first hypertrophy, and then softening).

Nervous Phenomena.

Are caused by the urea inducing toxic symptoms.

Headache, if constant, is a dangerous symptom, and of great importance, as it is apt to indicate the possibility of convulsions.

Drowsiness,
Stupor,
Nausea,
Vomiting,
Fever,
Convulsions,
Coma,
} may be developed if the poisoning be gradual.

Death results from the effects of urea upon the nervous system, or from some of the complications of uræmic poisoning.

Theories for Convulsions.

1. *Urea in excess* in the blood (which poisons the nerve-centres directly).

2. Œdema of the medulla (causing pressure upon it).

3. Urea becomes an active poison by being changed into ammonium carbonate (which causes irritation to the nerve-centres).

Uræmic convulsions usually come on in the manner herein indicated. Great nervous prostration will probably have previously existed, as well as headache and muscæ volitantes; sudden stiffening of the muscles; eyes, rolled up and fixed; head, turned to one side; teeth, tightly clenched; countenance, becomes livid; inspirations, long and deep; muscular twitchings; profound sleep; tongue, may be bitten, or, fall back on the epiglottis, closing the larynx, and thus produce death.

To determine the quantity of solid matter excreted daily.

Multiply the two last figures of the specific gravity of the urine by the number of ounces passed by the patient during the twenty-four hours, and the result will be the quantity of solid matter excreted

in the urine (in grains). Five hundred grains of urea being the normal quantity excreted daily in health.

It is estimated that from six hundred to one thousand and twenty grains of solid matter are excreted daily by a healthy individual; and 25 to 80 fluid ounces of urine passed daily.

CAUSES OF DEATH.

Death may be due to uræmia; meningitis; peri- and endo-carditis; or pneumonia.

COMPLICATIONS.

Cardiac hypertrophy may occur in the chronic form of Bright's disease, and is due to interference with the systemic circulation from long-continued excess of urea.

In the lungs and bronchi.

Pulmonary œdema—may develop in the first stage, or is one of the results of a general dropsy.

Pneumonia—if developing in connection with pulmonary œdema, and if albumen and casts be found in the urine, offers an unfavorable prognosis.

Bronchitis—This may occur as a complication of Bright's, with or without pulmonary œdema.

Fluid in the pleura, in the latter stages of Bright's disease, may occur as an evidence of serous inflammation, or of general anasarca.

Endocarditis—(from the irritating influence of poison) may exist as one of the serious complications of Bright's disease.

Meningitis—(as another serious complication) may be developed as a result of uræmic poisoning.

Subacute inflammation of the mucous membrane of the stomach (structural changes).

Amaurosis or neuro-retinitis (loss of sight).

Erysipelas—from poisoning of the nerve-centres, or from a defective nutrition of the tissues.

Gangrene—from pressure on the vessels by the œdema (cutting off the nutrition); from changes in the capillaries (which interfere with the circulation of the part); from

defective heart-power (causing poor nutrition); and from the direct effects of the poisoned condition of the blood.

DIFFERENTIAL DIAGNOSIS.

From typhus fever and the amyloid and cirrhotic forms of kidney disease.

PROGNOSIS.

Is bad as regards complete recovery. In the *first stage*, the prognosis varies with the character and intensity of the inflammatory process. In nearly every instance, the catarrhal or croupous forms may be recovered from. If with the croupous variety degeneration and desquamation go on slowly complete recovery is rare.

In the *second stage* (fatty) the chances for a complete recovery are very doubtful, if this stage has been reached by epithelial degeneration, and evidences of extensive obstruction in the tubules exist, the stage of granular degeneration and atrophy are sure to follow.

TREATMENT.

In the First or Inflammatory Stage.

1. *Eliminate the urea.*
2. *Remove the inflammatory products in the uriniferous tubules.*
3. *Counteract the effect of the urea on the nervous system.*

First, remove the exudation in the tubules (which develops degenerative inflammation). *Digitalis* must be given, as it increases the secretions, and does not stimulate the kidneys; it also increases the power of the heart's action, and overcomes the obstruction in the renal circulation, causing an increased flow through the Malpighian tufts in the upper portion of the uriniferous tubules. It should be given *in large doses. Inf. dig.*, ʒ ss. *every two hours* for twenty-four hours, and wait twelve hours, and watch it, then give smaller doses (ʒ i.), and stop as soon as you have good diuresis. *Dry cups* should be applied over the region of the kidney, in conjunction with *digitalis;* it draws the blood to the surface, but does not cause extravasation. Then apply warm poultices made of digitalis leaves. Temporary *hot bath* and *hydragogue cathartic* may be resorted to if the uræmic symptoms are urgent.

Oleum tiglii, (*oleum crotoni*) (but not after free diuresis has been obtained). In case of the following symptoms occurring, as headache, vertigo, restlessness, convulsions, and coma, the treatment for acute uræmia should be followed (see page 367). Then *actual cautery* may be applied over the lumbar region; it acts on the sympathetic nerves, and causes contraction of the renal blood-vessels. Depletion should only be used in the first stage of Bright's, and the form which follows pregnancy, surgery on genito-urinary tract, and exposure.

In the Second or Degenerative Stage.

Administer *digitalis* in moderate doses, and thus remove the fatty accumulations from the tubules. *Iron* should be given with a most nutritious diet. The patient should be fed on about half a pint to a pint of *milk* at a time, consuming about three to four quarts in the twenty-four hours. *Moderate stimulants*, such as wines, may be given. The patient must have the best hygienic surroundings, be kept in a well-ventilated department, with an uniform temperature, and the body be covered with flannel. Recovery may take place in about six weeks, if no tubular degenerative process commences.

In the Third or Atrophic Stage.

Diuretics are not required; a supporting plan of treatment is indicated. Food should be given in small quantities, and often, and let them have *cod-liver oil and iron*. *Wines* should be administered in moderation. *Cover the body with flannel*, and do not expose it to sudden changes in temperature; or send the patient to a warm climate. Should the urine become scanty, give two or three full doses of *digitalis*, to prevent acute tubular nephritic inflammation.

AMYLOID KIDNEY.

DEFINITION.

Is a non-inflammatory condition of the kidney, which commences as a deposit of amyloid material in the walls of the blood-

vessels. It is always chronic in its course, and usually invades several organs at the same time.

MORBID ANATOMY.

The minute arteries are first affected; then changes occur in the secreting tubes, or cells; and, finally, atrophy of the organ itself takes place. It has three stages.

1st Stage. Degeneration of the walls of the blood-vessels.

2d Stage. Changes in the blood-vessels and the uriniferous tubes.

3d Stage. Atrophy of the organ.

In the First Stage.—(Degeneration of the walls of the blood-vessels). The kidney is slightly increased in size and firmer than normal. The capsule is readily removed, and the surface is smooth and paler than normal.

On section.—If Lugol's solution of iodine be applied, with a clean camel's-hair brush, over the suspected portion, in a few seconds, if amyloid material be present, the affected part rapidly absorbs the iodine, and results in a dark-brown tint which is easily distinguished from the yellow stain of the non diseased tissue. There are glistening streaks along the course of the blood-vessels, and especially in dots corresponding to the Malpighian tufts. These glistening portions of the organ give the blue reaction to the iodine and the sulphuric acid tests.

Microscope.—Shows the changes to be most marked in the Malpighian tufts, and that the middle coat of the small arteries has undergone more or less alteration from a deposit of amyloid material, and are irregular in outline. This irregularity of outline is due to the amyloid deposit in the coats of the blood-vessels.

In the second stage. The kidney is very much increased in size, the capsule is adherent; and the surface pale and smooth and presents a stellate vascularity,

On section.—The increase in the size of the organ is seen to be due to an increased development in the cortical substance. The blood-vessels have undergone a waxy change, and the whole kidney has a waxy appearance.

The *microscope* shows the entire surface to have a shining, waxy appearance. The tubules in the medullary substance are dis-

tended with a substance similar to that found in the coats of the blood-vessels (but does not respond to the iodine test). This substance may be broken-down epithelium and fatty granules, or fibrinous material, like hyaline casts. The Malpighian capillaries are thickened, opaque, and glistening, and there is degeneration of the larger blood-vessels. The casts are chiefly in the large tubes of the pyramids.

In the Third Stage.—(Atrophy.)

On section.—The size of the kidney is diminished; the capsule adherent; the surface uneven, pale, and waxy. There is a diminution in the size of the kidney (due to a decrease of both the medullary and cortical substance). The Malpighian tufts are large and prominent. The small arteries are enlarged; their walls are impervious; and their circulation obstructed.

The *microscope* shows the tubules to be more or less atrophied, and their walls collapsed. The blood-vessels are thickened and irregular in outline. The iodine test decides the nature of the change.

ETIOLOGY.

Tertiary syphilis; prolonged suppuration (especially in diseases of the bone); suppurative diseases of the lungs (empyema, phthisis).

SYMPTOMS.

General emaciation (from some wasting disease).
Impaired mental faculties, and, possibly, insomnia.
Œdema of the feet at night.
Dyspnœa—on exertion (probably due to anæmia).
Complexion—pale and waxy (often with pigment in the eyelids).
A sense of fullness and weight in the abdomen.
Urine—increased; patient rises two or three times in the night to micturate.
General anasarca—may occur.
Perspiration—has an urinous odor.
Dyspepsia.
Nausea.
Vomiting.

Spleen—enlarged.
Liver—enlarged, with smooth, sharp edges (due to amyloid changes).
Cachexia.

CAUSES OF DEATH.

Death may occur from exhaustion (more frequently from amyloid degeneration of other organs); from diarrhœa (due to amyloid degeneration of the mucous membrane of the intestines); and from ascites (due to changes in the liver).

If dyspeptic symptoms do not yield to treatment, and you have also enlarged liver and spleen and diarrhœa, it is beyond a doubt that amyloid degeneration of the intestines has caused death.

The *principal symptoms* that have to be borne in mind *are* those in connection with the *urine*, the development of slight *dropsy*, and the absence of marked *nervous phenomena*.

Urine.

Increased in quantity—may reach one hundred ounces in twenty-four hours. Forty ounces in twenty-four hours is a small quantity. It is of a light color, like clear water, or slightly amber. Low specific gravity (1.005).
Albumen—not large in quantity (only a trace may often be detected); if large in quantity, it shows that tubular inflammation has occurred in a degenerated kidney.
Casts—large hyaline or granular, or both, but not abundant; epithelial or fatty casts may also be detected (showing inflammatory process of the tubules).

Dropsy.

Is never very marked.
Œdema—of the feet at night, probably due to changes in the liver.

Nervous Phenomena.

Rarely headache, convulsions, or coma.

DIFFERENTIAL DIAGNOSIS.

Is easy; for, if from the commencement of the disease there has been a copious secretion of urine, of low specific gravity, containing little albumen, and the symptoms of the disease have come on gradually in one who is suffering from an exhaustive

disease, you may expect to find amyloid kidneys. The probabilities are that the kidneys will be increased in size, if the liver and spleen are found to be enlarged.

PROGNOSIS.

The duration of life is uncertain. It usually takes years to establish the disease, but, when once established, is fatal.

TREATMENT.

If possible, remove the cause, as it occurs in the diseases of the bone, suppuration, etc.; therefore, remove the purulent accumulations. If occurring from tertiary syphilis, use antisyphilitic remedies, as *iodide of potassium* and *mercury*. *Mercury* should be given *in small doses, and often* (but do not produce ptyalism); but when debility exists, give *iodide of potassium* and *cod-liver oil*. *Iodide of iron* may be given in the form of *Blanchard's pills;* give one three times a day when taking food. Hydragogue cathartics and diuretics are of no service, excepting when the inflammatory form exist, then resort to dry cups, etc.

CIRRHOTIC KIDNEY.

SYNONYMS.

"Gin-drinkers' kidney;" granular kidney; contracted, or "hob-nailed" kidney.

DEFINITION.

Is due to an increase in the intertubular structure, and an atrophy of all the other structures.

STAGES.

1st Stage. *Enlargement*—due to an increase in the intertubular tissue (from connective-tissue cell-growth).

2d Stage. *Organization of this new tissue.*

3d Stage. *Atrophy*—due to contraction of this new tissue (producing degeneration, on account of pressure on the blood-vessels of the organ).

MORBID ANATOMY.

As the process progresses, there is shrinking of the kidney tissues. The kidney is diminished in size, the capsule thickened and adherent, and there is a prolongation of the connective-tissue into the cortical substance, causing removal of the structure when the capsule is torn off. Dilated veins may often be seen on the surface. The diminution in the size of the organ is due to a decrease in the cortical substance. The medullary substance retains the same appearance as normal, and is not diminished. There may be cysts near the surface on the cortical substance (if the tubes be obliterated, and the tufts of Malpighi continue to secrete).

In the first stage.—The *microscope* shows an increase in the connective-tissue between the Malpighian tufts and the uriniferous tubules. The kidney is slightly increased at first, and then shrinks; the Malpighian tufts are diminished; the uriniferous tubules are obliterated by contraction of the new connective-tissue; the capsule is thickened, firm, and fibrous. The arteries are hypertrophied and irregular; the tufts are brought nearer together by the increase in the connective-tissue, and are sometimes obliterated by cysts. The tubules may contain coagulated fibrin, which is indicated by hyaline casts in the urine. The changes are due to hyperæmia. Some say the inflammation is due to alcohol or gouty diathesis; if due to the latter, there is, in addition to the intertubular changes, a deposit of urates of soda in the cortical substance and apices of the cones.

The inflammatory process is the only cause of the anatomical changes in the organ.

ETIOLOGY.

The two most common causes are rheumatism and gout, producing gouty kidney (due to a change in the blood from these diseases); lead-poisoning, cancerous diathesis, and alcoholism, may produce, at the same time, cirrhosis, both of the liver and kidney.

SYMPTOMS.

Are obscure, and its approach is insidious.

Frequent micturition is usually present.

Urine—may not contain albumen nor casts.
Dropsy—may not occur.
Nervous symptoms may be the only well-defined ones.

The usual symptoms are

Debility.
Emaciation.
Dyspepsia.
Great thirst.
Urine—is very large in quantity.
Complexion—is of a dingy hue.
Œdema—slight, and usually confined to the lower extremities, after standing or walking. It is more marked at night than on rising in the morning.
Expression—care-worn. The patient often becomes fretful.
Nervousness.
Restlessness.
Insomnia (a prominent symptom in some cases).
Convulsions.
Coma—(for twenty-four hours or more before death).
Death.

The *principal symptoms* that have to be borne in mind *are* those in connection with the *urine*, the *dropsy*, and the *nervous phenomena.*

Urine.

Increased in quantity.
Low specific gravity (1.010).
Albumen—sometimes present, but inconstant.
Casts—usually large hyaline. They are often difficult to find, and may not be observed until three or four examinations have been made.

Dropsy.

Œdema—slight, and usually confined to the feet, after exercise or standing; but, if it become chronic in the feet, it is associated with general symptoms.
Ascites—may occur from changes in the liver.

Nervous Phenomena.

Headache—most prominent if associated with gout and rheumatism.
Vertigo. — High arterial pressure marked at radial pulse.
Temporary inability to speak may exist in some instances.
Amaurosis.
Deafness.
Numbness.
Neuralgic pains.
Cramps.
Delirium or insanity.
Insomnia—(great, and extending over a long period of time).
Paralysis—(temporary, and partial in the arm or leg).
Convulsions—(usually developed after some mental or physical exertion).
Coma—which may follow a condition of drowsiness, or delirium.
Tongue—dark-brown.
Pupils—dilated (if coma exist).
Death.

CAUSES OF DEATH.

Death usually takes place from convulsions, preceded by headache, and terminating in coma.

COMPLICATIONS.

Cardiac hypertrophy of the left ventricle—usually occurs from disease, atheroma, or calcareous degeneration of the walls of the large arteries.

If cardiac hypertrophy of the left ventricle exist, and the urine be abundant, of low specific gravity, and containing only a trace of albumen, it is sufficient to establish the diagnosis of cirrhotic kidney.

Mucous inflammations, especially bronchitis (of the chronic type), alternating with renal and gouty symptoms.

Amaurosis (of uræmic origin), called neuro-retinitis (first of one eye, then of the other).

Hemorrhage from serous and mucous surfaces, as well as in the substance of the organs. Cerebral hemorrhage is especially liable

to occur (cerebral apoplexy) from degeneration of the cerebral arteries, and an increased force of blood from an hypertrophied left ventricle (thus causing rupture of the capillaries).

DIFFERENTIAL DIAGNOSIS.

(1) From typhus fever, on account of the uræmic stupor and dry tongue. In typhus fever, however, there will be the eruption, and the quantity of urine passed may serve as a guide in the diagnosis.

(2) From gouty and rheumatic diathesis. In this there will be the history. The low specific gravity and profuse quantity of urine (which may contain albumen and casts) is sufficient to establish a diagnosis of cirrhotic kidney.

PROGNOSIS.

Is bad as regards recovery.

TREATMENT.

Hydragogue cathartics should be given when the disease is developed, in connection with cirrhosis of the liver. Resulting, as this disease usually does, from rheumatism or gout, and the patient suffering from headache, restlessness, nervous and tremulous affections, it is advisable to send this class of patients to the alkaline springs, as you would in cases of rheumatism or gout, and administer *bicarbonate of soda* and *potash in large doses.* Iron is not desirable, as it has too great an effect on the nervous system. *Strychnia* is preferable to iron, and should be given *in minute doses* for a long period. In many cases *cod-liver oil* should be combined *with the hypophosphate of soda.* These patients should reside permanently in a warm climate, all exciting causes of cirrhotic development should be avoided, and hygienic rules must be carefully observed. *Never give digitalis or iron.*

PYELITIS.

DEFINITION.

Is an inflammation of the mucous membrane of the pelvis and calices of the kidney. It may be acute or chronic in its course.

MORBID ANATOMY.

In the acute variety.—The mucous membrane of the pelvis is more or less congested. The surface is dotted with red spots (ecchymosis). The epithelial covering is more or less removed (may be entirely destroyed, or absent in patches). There is usually a muco-purulent secretion covering the surface of the mucous membrane. There may be a diphtheritic membranous exudation on the surface of the mucous membrane, which is due to a general diphtheria.

In the chronic variety.—The mucous membrane of the pelvis of the kidney is not only much thickened, but changed in color. It may be grayish-white or slate-colored, and traversed with dilated veins. The pelvis and infundibula are dilated. Pus is abundantly formed, and, if not obstructed, flows off with the urine; but, if there be obstruction, it accumulates and produces pyonephrosis. The accumulation of pus causes the papillæ to become flattened and obliterated. Then the pyramids are compressed, and, lastly, the cortical substance, both of which may eventually disappear on account of pressure. The sac so formed contains pus, broken-down material, or calcareous matter. If pyelitis be caused by obstruction from renal calculi, there may be ulceration of the mucous membrane, causing perforation of the pelvis and extravasation of urine into the adjacent tissues. The ureters may be the seat of disease, from an extension of pyelitis, and may be obstructed by pus, blood, and urinous material, producing a sac, like an abscess, which may be evacuated by an opening into the adjacent tissues. Inflammation may be established, and the pus may be discharged externally (by ulceration), or into the intestines. The accumulation may occasionally undergo absorption, transforming the ureters into a tendinous cord; in this case, the other kidney has to do double work.

ETIOLOGY.

Calculi, or any foreign body or substance in the pelvis of the kidney, may create it; and irritation (by decomposition of urine in the pelvis of the kidney) may prove an exciting cause. Urethral stricture or enlarged prostate (by causing retention, after-

ward cystitis, and inflammation of the mucous membrane, extending to the ureters) may create it. The lining of the ureters thus becomes thickened, and their calibre diminished, obstructing the flow from the kidney. The retention of urine in the bladder and the pelvis of the kidney causes a decomposition of urea into ammonium carbonate and water. The ammonium carbonate produces irritation, which induces inflammation of the lining membrane of the bladder and kidney (cystitis and pyelitis), or ammonæmia (from absorption of ammonia). Finally, blood-poisoning (of all forms); acute parenchymatous nephritis, with bloody urine; and overdoses of turpentine, cantharides, and other stimulating diuretics, may be mentioned as causes of pyelitis. It may be secondary to morbid processes, or to mechanical irritation, and occasionally follows exposure to cold or dampness.

SYMPTOMS.

Are modified by the causes which produce it, such as renal calculi, bladder diseases, etc., etc.

Acute Pyelitis.

Pain—in the back, always over one or both lumbar regions. It is usually aching in character, shooting down the course of the ureters. It occurs with micturition, and is incessant if micturition be excessive. It is also aggravated by exertion or change of position.

Urine—is usually acid, and contains blood mixed with mucus and epithelial-cells, pus-cells, and blood-globules (the epithelial-cells are of the type which normally exist in the pelvis and infundibula). Epithelial-cells denote an early stage of the disease. Specific gravity 1.025 to 1.030.

Is ammoniacal in the advanced stages, and often contains albumen.

If there be obstruction, the urine from the other kidney is normal. If the obstruction be removed, the urine may suddenly become purulent, and continue to be so.

Chronic Pyelitis.

Tumor—in the lumbar region.

Bulging between the crest of the ilium and the false ribs.

Ureters—completely obstructed.

Accumulation of fluid in the pelvis of the kidney.

Palpation—shows deep-seated fluctuation. The tumor is painful and tender on pressure.

Percussion—elicits a dullness whose limits depend on the outline of the tumor.

DIFFERENTIAL DIAGNOSIS.

From cystitis and peri-nephritic abscess.

In pyelitis, associated with chronic cystitis, or enlarged prostate, or stricture (should the pus be large in quantity, the urine slightly acid, the loin painful, and constant febrile movement and emaciation exist), it indicates chronic pyelitis.

In acute Pyelitis.

Urine—is acid, and contains epithelial cells of the pelvis and infundibula; also blood-globules and mucus.

If there be epithelial-cells and pus, it indicates an advanced stage.

In Pyo-nephrosis.

Urine—is acid, and contains pus in large quantities.

Pain—exists in the lumbar region.

Tumor—may be detected at the seat of the pain, which gradually increases. It may suddenly disappear, when copious flow of pus ensues, which affords great relief to the patient.

This tumor may be mistaken for hydro-nephrosis, hydatid cyst, and peri-nephritic abscess.

Pyelitis.

Is an inflammation of the pelvis and calices of the kidney.

Produced by stone in the pelvis of the kidney, or inflammation extending upward from the bladder, or the use of irritant drugs.

Urine—is acid, and contains blood, pus, and epithelium of the pelvis and infundibula.

No tumor exists.

Chills and high temperature may be developed.

No pain on motion is present.

Aspiration—gives negative results.

Peri-nephritic Abscess.

Is a suppuration of the connective-tissue surrounding the kidney.

A traumatic origin can usually be discovered.

Urinary symptoms — are negative (unless perforation take place).

Tumor—may occur and point externally.

Temperature—not so high as in pyelitis.

Pain—can be detected on motion of the leg and thigh upon the trunk.

Aspiration—by aspirating at the extremity of the last floating rib, pus may be drawn off.

PROGNOSIS.

If it be the simple uncomplicated catarrhal form, the prognosis is not unfavorable. If both kidneys be affected and there be purulent discharge, the prognosis is bad. If it be secondary to an enlarged prostate or to an extension of a chronic cystitis, urethral stricture, or cancer, the prognosis is hopeless. If it occur with calculi or hydatids, the prognosis is grave. If there should be pyo-nephrosis and the pus escape externally or into the intestines, recovery is possible; but if there should be pyo-nephrosis and the pelvis bursts into the peritoneal or thoracic cavities, the result is usually fatal.

TREATMENT.

If of an *acute type* and the fever be considerable, pain in the lumbar region severe, and bloody urine, *wet cups* should be freely applied to the loins, followed by *hot baths* and a sufficiently large *hypodermic of morphine* to relieve all pain. The patient should be kept in bed and drink freely of alkaline fluids.

In *chronic pyelitis.*—*Astringents* (tannic acid) should be given if pus be abundant. *Cod-liver oil* and *quinine* should be administered with a nutritious but not stimulating diet, and prolonged use of alkaline beverages. *Aspiration* should be resorted to, if the tumor be near the surface, and, should the case require it, a free permanent opening should be left.

HYDRO-NEPHROSIS.

SYNONYMS.

Dropsy of the kidney; renal dilatation.

DEFINITION.

Is a non-inflammatory affection, due to a collection of urine in the pelvis and infundibula of the kidney from obstruction; compressing and causing an atrophy of the renal substance and converting the kidney into a sac or pouch. This dilatation may affect the ureter and pelvis, or only the pelvis.

MORBID ANATOMY.

The papillæ become flattened, hardened, shrunken, and disappear. The renal substance gradually diminishes from pressure, becomes tough and resistant, and, in extreme cases, the kidney becomes a multilocular cyst, sometimes as large as a child's head, and the ureters the size of the small intestine, with thickened and convoluted walls. The contents of these cysts is variable. Urine, which is usually more watery than normal, containing more or less urinary salts and also blood, pus, epithelium, and albumen, is generally found.

ETIOLOGY.

From closure of the ureter by an external tumor, cancer, impaction of calculi or mucus, or by inflammation causing adhesion of the walls of the ureters; from stricture of the ureters; from a congenital valve at the upper end of the ureters; from an abnormal course of the renal artery, which may be wound round the ureter like a cord; from dilatation of the ureter; from an impeding discharge which may completely close the papillæ and produce entire suppression of the urinary secretion.

SYMPTOMS.

Tumor—detected in the lumbar region, which is of slow development, painless, and fluctuant. In occasional instances, there

is a rapid subsidence of the tumor and a large discharge of fluid from the urethra and bladder (diagnostic).

Rupture of these cysts is uncommon on account of hypertrophy of the tunics; the cysts may be of small size, or occasionally large enough to contain gallons of fluid.

DIFFERENTIAL DIAGNOSIS.

From ovarian cyst; ascites; hydatids; and pyo-nephrosis.

Between hydro-nephrosis and ovarian cysts:

In *hydro-nephrosis*—there is the presence of the colon in front of the swelling, and an absence of tympanitic percussion in the lumbar region.

Between hydro-nephrosis and ascites:

In *hydro-nephrosis*—there is no dullness in the lumbar regions, neither is the line of dullness affected by change in the position of the patient. No evidence of portal obstruction can be detected.

Between hydro-nephrosis and hydatids:

Unless hydatids be found in the urine it is impossible to make a diagnosis.

Between hydro-nephrosis and pyo-nephrosis:

In *hydro-nephrosis*—the urine is non-purulent and there is no suppuration. The constitutional symptoms are not so severe as in pyo-nephrosis.

PROGNOSIS.

When one kidney is affected, the prognosis is favorable, as spontaneous evacuations may occur; but when both kidneys are affected, the prognosis is bad, as uræmic symptoms will develop and death ensue.

TREATMENT.

Evacuate the tumor by careful *manipulation*, or by *aspiration* at the extremity of the last floating rib. Medical treatment is of no avail.

RENAL CALCULI.

These may be formed in the tubes of the pyramids, in the cortical substance, or in the pelvis. They may occur at any age; from the new-born infant to very old age, or even in the fœtus.

MORBID ANATOMY.

In the aged, the formation of calculi is always associated with a gouty diathesis (as deposits of urate of soda in the kidneys, in the pyramids, and cortical substance). They may also occur in connection with diseases of the bone (as carbonate and phosphate of lime in the tubes and pyramids). Cysts may be developed from permanent impaction, or the calculi may be washed down the tubes by the urine, and lodged in the pelvis. They may obstruct the ureters and may also tend to excite pyelitis, abscess, parenchymatous nephritis, pyo-nephrosis, or hydro-nephrosis. The nuclei of the calculus may be pus, blood, epithelium, grains of pigment, etc.

ETIOLOGY.

In children, they are usually of the uric acid variety. *In adults*, they are generally composed of lime and triple phosphates (with some nuclei, as above mentioned, for a starting-point).

SYMPTOMS.

These are aggravated by violent exercise and jolting, as in riding, horse-back riding, etc. They may last from a few hours to a few days, or the calculi may pass into the bladder and remain there, or they may become encysted and cease to irritate. A large portion of the kidney may be sometimes destroyed or atrophied as the result of a calculus.

Renal colic—due to the passage of calculus along the ureters; it is accompanied by the following symptoms:

Pain—sudden and intense in the region of the kidney of the affected side, radiating to the bladder, testis, inside of the

thighs, and to the end of the penis. It is so intense that the patient often shrieks violently and rolls about.

Vomiting—violent and frequent.

Testicle—retracted on the affected side.

Countenance—pale, and covered with profuse perspiration; great anxiety is manifested in the face.

Pulse—small.

Extremities—cool.

Fever—may occur, but is not usually present.

Urine—may be suppressed or scanty in amount, high-colored, bloody, and discharged in drops. It often causes a painful burning sensation during micturition, and may contain pus, blood, or epithelium.

DIFFERENTIAL DIAGNOSIS.

Renal colic may be confounded with neuralgia of the lower intercostal and abdominal nerves, but the urine containing pus, blood, and epithelium decides the question of the presence of renal calculi. It may be mistaken for blood-clots, or hydatids in the ureters; for spasm of the ureters; and confounded with hepatic colic (especially if the right kidney be attacked).

PROGNOSIS.

Is good, unless there be impaction and obstruction to the escape of urine or of the calculus itself.

TREATMENT.

Is the same as in pyelitis and pyo-nephritis. The paroxysm may be relieved by the free administration of *opium*, warm baths, and hot poultices to the loins and abdomen. If the pain be intense and the vomiting constant, *inhalations of chloroform* will frequently give speedy and permanent relief. Change of position and manipulation along the ureters and drinking something containing carbonic acid will often aid in dislodging the calculus.

NEW-GROWTHS OF THE KIDNEY.

These comprise (1) cancer, which is the only one that is of any clinical importance; (2) tumors in the intertubular tissue; (3)

syphilitic gummata; (4) fibroma in the pyramids; (5) lipoma, which are sometimes developed around the capsule of the kidney, and in the pelvis of an atrophied kidney, or in the cortical substance beneath the capsule, and (6) tubercles (small miliary, gray, or cheesy), which are met with only in connection with general tuberculosis.

RENAL CANCER.

May be a primary or secondary affection and may occur in both kidneys.

MORBID ANATOMY.

These deposits are usually of the medullary variety, developing in the form of circumscribed nodules in the cortical substances. They may become so large as to fill the whole abdominal cavity. They are often associated with cancer of the testicle.

ETIOLOGY.

Depends upon either hereditary taint or some type of local infection.

SYMPTOMS.

Rational.

Emaciation—gradual.

Pain—probably none (confined to the lumbar regions, if present).

Urine—probably no change, but there may be hæmaturia and albumen; when hæmaturia is present, it is constant.

Complexion—a peculiar waxy appearance may often be detected.

Physical.

Palpation.—When it attains a large size it can be felt through the abdominal walls. Its form and immobility prove it not to be an enlargement of the liver or spleen.

CAUSES OF DEATH.

Death may result from exhaustion; hemorrhage; acute parenchymatous nephritis; or from invasion from other organs.

PROGNOSIS.

Is bad.

TREATMENT.

Is palliative, and all that can be done is to relieve the symptoms and sustain the patient as much as possible.

ADDISON'S DISEASE.

DEFINITION.

Is a chronic caseous degeneration of the supra-renal cap sules, attended by a peculiar *dark bronzed appearance* of the skin and atrophy of the neighboring plexus of nerves.

MORBID ANATOMY.

The capsule is swollen, thickened, dark in color, and studded with extravasations of blood. It is firm in consistence, and its surface is irregular. It presents a grayish-white or whitish homogeneous substance, with caseous collections, and calcareous deposits. These caseous masses are composed of débris and cellular elements in a state of decay or degeneration; or crystals of cholesterin may often be found. The pigmentary deposit is in the lower layer of the rete Malpighi (seen by the microscope).

ETIOLOGY

Is unknown. It forms one of the symptoms of general tuberculosis, cancer, and amyloid and fatty degeneration.

SYMPTOMS.

Skin—becomes gradually dark.
Roots of the nails remain white.
Palms of the hands and soles of the feet are spotted.
Sclerotic of the eye—is of a pearly hue.
Black spots appear on the mouth and lips.
Extreme debility and dementia are often present.
Pain—in the back and epigastrium usually exists.
Dyspepsia.
Vomiting.
Diarrhœa—obstinate in character.
Epileptic convulsions—may occur in some cases.

Temperature—usually normal.
Heart-beat and pulse—at first accelerated, but, toward the end, become weak and feeble.
Death.

TREATMENT.

Good nourishment and care is all that can be suggested, as it usually terminates in death in about four years at the most.

SURGICAL KIDNEY.

Is that type of kidney which is liable to be a sequel to operations on the genito-urinary tract, especially following the treatment of deep-seated stricture and operation for stone.

It is sometimes called "acute interstitial nephritis," because, in extreme cases, suppuration of the intertubular structure takes place, forming scattered abscesses throughout the kidney, which vary in size from a pin's head upward.

In the majority of instances, intense renal engorgement is the only lesion detected unless evidences of previous diseases exist.

Its chief symptom is *suppression of urine;* and, in fatal cases, both kidneys are affected. It is ushered in by a marked chill or rigors, within the first two days after the operation.

TREATMENT.

Is both preventive and curative.

Preventive.—Before the operation the patient should be confined to his bed for several days; he should have hot hip-baths; and be made to drink freely of diluent beverages. *Quinine* should be given *in large doses* and (Long's treatment) *Fleming's tincture of aconite,* gtt. ij. doses both before and after the operation.

Curative.—Inf. digitalis and citrate of potash; hot applications; wet cupping; blisters and actual cautery over the lumbar region; hot-air bath; hydragogue cathartics; morphia hypodermically.

CHOREA.

CHOREA.

SYNONYMS.

St. Vitus' Dance. St. Guy's Dance. It has been termed "insanity of the muscles."

DEFINITION.

Is a disease of a purely motor neurosis, and characterized by irregular clonic spasms of groups of muscles under the influence of the will, usually occurring on one side of the body, but which may become general.

MORBID ANATOMY.

The pathology, notwithstanding the numerous autopsies that have been made in connection with this disease, is admitted to be obscure, for it has never been discovered where the seat of the disease occurs in the central nervous organs, nor what are the morbid processes in the parts affected. On account of its unilateral origin and unilateral tendency, the disease is supposed to have commenced either in the crus cerebri, or in the corresponding corpus striatum and optic thalamus or, in the cerebral hemisphere. The majority of authors are totally averse to such opinions, in consequence of other parts which are supplied by these centers being frequently very little, if at all, affected during the course of this disease, and, furthermore, its occurring as a sequela to other diseases, as will be seen in its etiology.

Dr. Dickinson, from his results of careful autopsies of several fatal cases of this disease, in a valuable paper read before the Medico-Chirurgical Society, has come to the conclusion that chorea depends "on a widely-spread hyperœmia of the nervous centers, not due to any mechanical mischance, but produced by causes mainly of two kinds—one being the rheumatic condition, the other comprising various forms of irritation, mental and reflex, belonging especially to the nervous system, and in consequence of the vascular changes, sclerosis is induced in the tissues,

which surround the vessels, thereby explaining the rigidity of the limbs, permanent paralysis, and wasting of the muscles, which complicate or supervene upon this disease."

ETIOLOGY.

It occurs principally in female children at the period of second dentition and puberty. It rarely occurs before the sixth or after the fifteenth year of life. It may be hereditary. It may even be brought on through children mimicking others who have the disease (this frequently occurring in large boarding schools). It may be due to an irritation of peripheral portions of the nervous system. From blows; falls; wounding a nerve; uterine disorders; painful menstruation; pregnancy (rarely before the end of the second month); gastric or intestinal affections (by acting as the exciting cause of perverted muscular movements); strong mental emotions (fright, fear); over-fatigue; over-study; scarlet fever; rheumatism; bad hygienic surroundings; anæmia and hydrœmia seem to augment the tendency to this disease; it may occur in cardiac complications (rare) or spasmodic action of the papillary muscles; worms; onanism; hysteria.

SYMPTOMS.

Usually come on insiduously. The child becomes mopish, avoids its companions, takes no interest in games or pleasure, cannot commit lessons to memory, or recollect readily.

Real or apparent mental deficiency.

Fretfulness or capriciousness.

Suspicion or timidity.

Impairment of mental function.

Restlessness or fidgetiness.

Clumsy movements.

Inability to hold things steadily in the hand, and inability to write or work.

Choreic Movements upon one side (usually) either in the face, eye, tongue, head and neck, hitching of the shoulders, hand, arm (or both hand and arm), sometimes in the leg, may extend to the whole side of the body, and may become very characteristic, consisting in sudden impulsive movements following

each other at irregular intervals, involving first one group of muscles, then another, then one part of the body, and may ultimately involve the whole muscular system. These symptoms become greatly aggravated whenever the patient endeavors to accomplish any voluntary movement (as in drinking a glass of water), or when anything occurs to excite him, or when he knows he is being watched.

Difficulty of speech (due to convulsive action of the lips and tongue, or a spasmodic affection of the larynx and the respiratory muscles).

The lower limbs are dragged in walking.

Mastication and deglutition may be affected.

Pupils of the eye may be greatly dilated, and the sclerotic coat whiter than normal.

Respiration may be irregular, jerky and noisy in consequence of the diaphragm being affected.

Indigestion (may occur).

Sphincters of the rectum and bladder may be affected (in severe cases).

Constipation (may occur).

Impairment of menstrual functions.

Locomotion or use of the hands may become impossible (in severe cases).

Impairment of muscular strength.

Paresis (may occur when unilateral).

Hemiplegia or paraplegia may replace the convulsive movements.

Impairment of sensation.

Eyesight may fail.

Appetite may be bad, capricious, or even fail.

Cardiac palpitation (may occur), or even a murmur may be heard, which is attributed by Sir William Jenner "to an irregular action of the muscular apparatus of the mitral valve, and sometimes to an irregular contraction of the heart itself," but more probably as the result of anæmia.

Intellect develops slowly and is enfeebled as long as the disease lasts.

Sleep is disturbed, or insomnia (may occur in severe cases after assuming the form of nightmare).
Urine contains an excess of phosphates.

PROGNOSIS.

Is rarely fatal, but is not of short duration, for although being of a very acute nature, usually lasts from one to several months (due to successive relapses), the patient remaining subject to nervous disorders, and may in after years be followed by epilepsy, insanity, or muscular atrophy. When fatal (in severe cases), death is usually due to asthenia, erysipelas, to cardiac complications; or, it may result from extreme agitation; nervous exhaustion produced by excessive jactitations, exaggerated by sleeplessness, or cerebral rheumatism.

Complications—Endocarditis and pericarditis, which usually precede the disease (being due to rheumatism)—mental derangements.

DIFFERENTIAL DIAGNOSIS.

From spasms of acute cerebral diseases; from epilepsy; from tetanus; from paralysis agitans; from convulsive tremor; from mercurial tremor; from facial spasm; from writers' cramp; from salaam convulsions; from locomotor ataxia.

TREATMENT.

Cold baths have been advocated, as also river and sea-bathing, but on account of their great tendency to produce rheumatism in children, they have been abandoned by their greatest advocates. These methods have been replaced by dipping the patient in baths (two or three times at first), at a temperature about 75° F., gradually lowering the temperature every day.

Sulphur baths have been also recommended, prepared in the following manner, from ½ to 1½ ounces of sulphuret of potassium is dissolved in 12 gallons of water, at a temperature of 86° F. These baths must be repeated daily with the greatest regularity, in which the patient is to spend about one hour at a time, but where rheumatism threatens this treatment must not be resorted to.

Tonics and the preparations of *iron* are indicated when the disease is due to chlorosis.

Arsenic (especially Fowler's solution) is largely prescribed, in consequence of its possessing the property of causing general excitation, and especially, increased vigor of the lower extremities. I have seen very excellent results by combining the citrate of quinine and iron with this drug. ℞ Lig. pot. arsen : ʒ i–ii Ferri et quin. cit ʒ i–ii. Glycerinœ et aquæ aa ℥ i.–ʒ i t. i. d. after meals—gradually increasing the doses of the arsenic every week. Should the disease be due to struma, *iodine* and *iodide of potassium* are indicated.

Tartar Emetic in large doses, in consequence of its sedative action on the nervous system, had its advocates, but was like many other remedies abandoned. Dr. Roger, in an article published in the "Union Medicale," July, 1858, gives the following rules for its administration : "On the first day, doses of 4 to 5 grains in the 24 hours, which is doubled on the second day, and trebled on the third day, and then a period of rest for 3 to 5 days. If the disease persist, or the convulsive movements merely diminish in violence, the drug is again administered for another three days, beginning with the same dose as on the first day, plus an additional grain, then an additional rest. If, after this period, the disease recur, the same plan is adopted. So that, if the first dose, given on the first day of the first series, be 4 grains, that on the first day of the second series 5 grains, and on the first day of the third series 6 grains, and on the third day of the third series 18 grains." Antispasmodics and narcotics such as *assafœtida*, *camphor*, *musk*, *valerian*, etc., have been recommended, put aside, and tried again.

Cannabis Indica has been greatly praised by Dr. Corrigan, of London, who commences the treatment by giving 5 minims of the tincture three times a day for a week or ten days, then gradually increasing the dose to 25 min. three times a day. This drug possessing, as it does, an alterative action on the nervous system, should be used in obstinate cases, especially where narcotics are indicated, in order to prevent dangerous complications.

Opium finds a strong advocate in Trousseau, where the disease

is complicated by insomnia, which gradually exhausts the patient's strength, the *sulphate of morphia* being preferred, giving it in grain doses every four hours. He increased the dose until 30 grains had been administered in the twenty-four hours, when he obtained complete recovery. In very severe cases, when the movements become beyond control, *chloroform* should be administered.

Sulphate of Zinc, from 2 to 3 grains three times a day, dissolved in a sufficient quantity of water to prevent gastric irritation, was at one time used by Dr. Hammond, of this city, but he now prefers the *bromide*, beginning with ten drops, three times a day, increasing the dose till the symptoms begin to disappear, and then decreasing the dose accordingly.

Gymnastic Exercises have their advocates among many practitioners, which, when properly carried out have many advantages. Whenever the vertebræ are sensitive to pressure, *leeches* and *cupping* have been applied to the side of the spine, followed by counter-irritants.

Hygienic Measures, as they occupy a prominent part, should not be forgotten in this disease. The patient should sleep in a well-ventilated room; nutritious and tonic food should be taken at regular intervals; *cod liver oil*, if there be great debility; open air exercise, without producing fatigue, in order to facilitate the organic movements of repair, thus preventing recurrence of the disease; the bowels should be properly regulated; cold bathing and swimming may be indulged in *in moderation*. When the disease assumes the severe type it is advisable to separate the patient from others, on account of the annoyance, and because the affection is apt to extend from one to the other from sympathetic irritation.

PRESCRIPTIONS.

PRESCRIPTIONS.

ALKALINE MIXTURE FOR NURSING CHILDREN.

℞ Pot. carb.....gr. ij.
Ol. cajuput.....♏ i.
Aq. anethi..... ʒ ij.

M. Sig. three or four times a day.

ASCARIDES.

℞ Quassiæ..... ʒ ij.
Ac. salicyl.....gr. x.
Aquæ... .....O.i.

M. Sig. to be used as an enema.

ASTHMA.

℞ Fl. ext. ergotæ,
Tr. hyos..... .....āā ʒ i.
Hoff. anodyne..... ʒ ij.

M. Sig. ʒ i. every half-hour till relieved, then every 2 hours for the next 24 hours; then give

℞ Mist. glycyrrh..... ʒ iij.
Tr. opii camph..... ʒ i.
Ammon. mur .....gr. lx.

M. Sig. ʒ i. t. i. d.

If it become severe, paint the chest with

℞ Canth. collod. sol. over a space 3 inches by 8 inches and give

℞ Hyd. chlor. mit.....gr. x.
Pulv. Jalapæ.....gr. xv.

Or,

℞ Pot. iodidi,
Tr. bella.....āā ʒ i.
Liq. pot. arsen.....♏ xl.
Spts. ether. co..... ʒ iss.
Elixir simplicis..... ʒ ij.
Aq. cinnamon..... ʒ vi.

M. Sig. ʒ iv. t. i. d. after meals.

ASTHMA (HAY).

℞ Pot. iodidi..... ʒ i.
Liq. pot. arsen..... ʒ i.
Aquæ..... ʒ iv.

M. Sig. ʒ i. every 4 hours.

Or,

℞ Liq. pot. arsen..... .... ʒ iij.
Tr. bella ..... ʒ ix.

M. Sig. gtt. xij. after meals, and increase in 3 days to xvi., and after 10 days to gtt. xx.

ASTHMA (IN PREGNANCY).

℞ Pot. brom ..... ʒ iij.
Chioral hydrat..... ʒ ss.
Aquæ..... ʒ i.

M. Sig. ʒ j. to be used as required.

Or,

℞ Pot iodidi..... ʒ i.
Iodini.....gr. i.
Tr. gent. co ..... ʒ i.
Aquæ ..... ʒ ij.

M. Sig. ʒ i. t. i. d.

ASTHMA (SPASMODIC).

℞ Pot. iodidi .....gr. xv.-xxx.
Every 2 or 3 hours.

ASTHMA (IN A CHILD).

First, for sneezing, keep the bowels open with a Gregory's powder, and give

Pulv. Dov.,
Quin. sulph.....āā gr. viij.

Also,

Spts. nit. dulc.,
Tr. Scillæ,
Tr. opii camph.....āā ʒ i.

Sig. ʒ i. every hour till relieved.

ASTHMA (With Emphysema and Chronic Bronchitis).

℞ Syr. prun. virg..... ʒiv.
Hoff. anodyne..... ʒss.
Pot. iodidi..... ..... ʒij.
Tr. bella..... ʒij.-iv.

M. Sig. ʒi. t. i. d. and send the patient to a warm climate.

ANÆMIA (In Children).

℞ Ferri cit..... ʒij.
Aq. aurant..... ʒvss.
Syr. simplic..... ..... ʒss.

M. Sig. from a teaspoonful to a tablespoonful before or after each meal.

ANÆMIA.

℞ Tr. ferri perchlor.,
Acid. acet. dil .....āā ♏ xx.
Syrupi ..... ʒij.
Liq. ammon. acet..... ʒvi.

M. Sig. ʒij.-iv. every three hours.

ACNE.

℞ Tr. sap. vir..... ...ʒiij.
Ac. carbol..... ʒss.
Sp. vini rect..... ....ad ʒiv.

M. Sig. to be used at night. In the morning the parts should be washed with warm water and then rubbed down with

Ungt. hydrarg, ammon.,
Zinci oxidi.....āā ʒss.

ANEURISM.

℞ Pot. iodidi .....gr. xv.-ʒss.
Sig. take this amount t. i. d.

APERIENT.

℞ Fl. ext. rhei..... ..♏ xl.
Fl. ext. ipecac.....♏ iij.
Sodii bicarb..... ℈ij.
Aq. menth. pip.....ad ʒij.

AMENORRHŒA.

℞ Ferri. arsen.....gr. v.
Ergotinæ (aq. ext.)..... ʒss.
M. et ft. pil. xxx.
Sig. One night and morning.

Or (Tonic).

℞ Sulph. strych .....gr. i.
Ferri. pyrophos.,
* Quin. sulph .....āā ʒi.
Ac. phos. dil.,
Syr zingiber.....āā ʒij.
M. Sig. ʒi. t. i. d.

ANGINA PECTORIS.

℞ Ammon. valer.....gr. v.-x.
Chlor. ammon.....gr. xxx.
Sig. take immediately.

Or,

℞ Tr. rhei et sennæ..... ʒiss.
Syr. zingib..... ...ʒiij.
Tr. opii..... ʒi.

M. Sig. ʒi. in hot water immediately.

ANUS (Itching of).

℞ Pot. brom.,
Chlor. hydrat.....āā gr. x.-xv.
Aquæ..... ʒi.

M. Sig. To be taken at bed-time.

Also,

℞ Sodii bibor..... ʒij.
Morph. mur .....gr. xvi.
Ac. hydrocyan. dil.... ʒss.
Glycerinæ..... ʒij.
Aquæ. ....ad ʒviij.

M. Sig. to be applied to the affected part.

Or,

℞ Chloroformi..... ʒij.
Glycerinæ..... ʒss.
Cerat. simp..... ʒiss.
M. et ft. ungt. Apply locally.

(* Tr. hydrast. may be substituted.)

ARTHRITIS (CHRONIC).

℞ Lithii brom..... ʒ iij.
Syr. zingiber..... ʒ ss.
Aquæ..... ʒ iss.
M. Sig. ʒ i. t. i. d.

Or,

℞ Lithii carbonat..... ʒ j.
Ac. citrici..... ʒ ij.
Aquæ..... ʒ ij.
M. Sig. ʒ i. t. i. d.

ARTHRITIS.

℞ Ol. sassafras..... ʒ ij.
Aq. ammon..... ʒ iv.
Lin. sap. camph..... ʒ iij.
M. ft. liniment (for ext. application)

APHTHÆ OF PHTHISIS.

℞ Quin. sulph.....gr. i.
Olei piperis nig.....gtt. i.
Aquæ..... ʒ i.

Sig. apply with a brush or rinse out the mouth.

BLADDER (INFLAM. OF).

℞ Tr. bella..... ʒ i.
Fl. ext. buchu..... ʒ i.
Liq. pot. arsen ..... ʒ ss.
Syr. simp..... ʒ iss.
Tr. opii camph..... ʒ vi.
Aquæ.....ad ʒ vi.

M. Sig. ʒ i. every 3 hours. (Tr. opii camph. should not be included when blood exists in the urine.)

Or,

℞ Pot. brom..... ʒ iij.
Chloral hydrat..... ʒ iss.
Syr. rub. idæi,
Aquæ..... āā ʒ i.

M. Sig. ʒ i. every half-hour till relieved (and apply hot fomentations).

BLADDER (IRRITATION OF).

℞ Pulv. opii .....gr. ij.
Ol. theobro.....q. s.
M. ft. suppos. 1.

Or,

℞ Acid. benzoic..... ʒ ij.
Aq. cinnam..... ʒ vi.
M. Sig. a tablespoonful t. i. d.

BREAST LOTION.

℞ Ext. bella..... ʒ i.-ij.
Pulv. camph..... ʒ i.
Glycerinæ ..... ʒ ij.
Chloroformi ..... ʒ ss.
Ol. menth. pip..... ʒ i.
M. Sig. apply locally.

BILIARY CALCULI.

℞ Phos. sodæ..... ʒ i.
Sig. take this t. i. d.

Or,

℞ Bismuthi,
Inf. calumb.....āā ʒ ij.
Hydrat chloral ..... ʒ ss.
Syr. rub. idæi,
Aquæ.....āā ʒ ij.
M. Sig. ʒ i. t. i. d.

BRONCHITIS.

℞ Pot. nit..... ʒ ij.
Pulv. sacch. albi..... ʒ ij.
M. et ft. chart. xij.
Sig. one every two or three hours.

BRONCHITIS (ACUTE).

℞ Vini ipecac..... ʒ ij.
Liq. pot. cit..... ʒ iv.
Tr. opii camph.,
Syr. acaciæ.....āā ʒ i.
M. Sig. ʒ ij. t. i. d.

Or,

℞ Ant. et pot. tart.,
Morph. acet.....āā gr. i.
Aquæ destil..... ʒ iv.
M. Sig. ʒ i. every hour.

BRONCHITIS (AFTER LOOSENING OF COUGH).

℞ Syr. scillæ..... ʒ iij.
Tr. opii camph..... ʒ i.
M. Sig. ʒ i. t. i. d. or ʒ ij. at night.

BRONCHITIS (CHRONIC).

℞ Ammon. chlor..... ʒ ij.
Mist. glycyrrh. co..... ʒ iij.
M. Sig. dessertspoonful t. i. d.

Or,

℞ Ext. eucalypt..... ʒ i.
Ammon. mur.,
Ext. glycyrrh..... āā ʒ ij.
Syr. tolu..... ʒ iij.
M. Sig. ʒ i. every two hours.

Or,

℞ Ammon. chlor..... ʒ iij.
Muc. acaciæ.....fl. ʒ iv.
M. Sig. a tablespoonful four times a a day.

BRONCHITIS (WITH DRY COUGH).

℞ Antimon. tart..... .....gr. i.
Syr. scillæ ..... ʒ iv.
M. Sig. a tablespoonful every 3 hours.

BRONCHITIS (Chronic) (With Asthma and Emphysema).

℞ Syr. prun. virg..... ʒ iv.
Spt. œther. comp. (Hoff.). ʒ ss.
Pot. iodidi..... ʒ ij.
Tr. bella..... ʒ ij.-iv.

M. Sig. ʒ i. t. i. d. and send the patient to a warm climate.

BRONCHITIS (Acute in Children).

℞ Tr. ver. vir..... ♏ij.
Syr. scillæ comp..... ʒ ij.
Syr. balsam. tolu..... ʒ xiv.

M. Sig. ʒ i. every 2 or 3 hours to a child 5 years old, in first stage of the disease.

BRONCHITIS (In Infants).

℞ Syr. senegæ..... ʒ i.
Syr. tolu..... ʒ ij.
Ammon. mur..... gr. x.

M. Sig. ʒ i. (small) every 3 hours.

BRONCHITIS (2d Stage).

℞ Sulph. morph..... gr. i.
Syr. bals. tolu.,
Syr. prun. virg..... āā ʒ i.

M. Sig. ʒ i. t. i. d., and if there be night-sweats add morphine gr. 1-60.

Or,

℞ Sulph. morph..... gr. i.
Vin. antimon..... ʒ ij.
Syr. tolu..... ʒ iss.
Aquæ..... ʒ iij.

M. Sig. ʒ i. t. i. d.

CALCULI (Biliary).

℞ Bismuthi,
Inf. calumb.....āā ʒ ij.
Hydrat chloral.....ʒ ss.
Syr. rub. idæi,
Aquæ.....āā ʒ ij.

CARDIAC HYPERTROPHY (Without Valvular Lesion).

℞ Fl. ext. ergotæ.....ʒ iiiss.
Tr. digit.....ʒ ss.
M. Sig. ʒ i. t. i. d.

CATAMENIA (Stopped).

℞ Pot. brom.....ʒ iv.
Aquæ.....ʒ ij.
M. Sig. ʒ i. t. i. d.

CHANCRE.

℞ Hyd. chlor. mit.....ʒ i.
Liq. calcis.....ʒ iv.
M. et ft. lotio. Sig. apply on lint.
Or,
℞ Hyd. chlor. corr.....gr. xvi.
Liq. calcis.....ʒ viij.
M. et ft. lotio. Sig. apply on lint.

CLAP.

℞ Tinct. hydras.....ʒ ij.
Aquæ.....ʒ viij.
M. ft. lotio, inject 3 or 4 times daily.
Or,
℞ Fl. ext. hydras.,
Glycerinæ.....āā ʒ ij.
M. et ft. lotio for injection, t. i. d.
And,
℞ Tinct. gelsemii.....ʒ i.
Aquæ.....ʒ iij.
M. Sig. ʒ i. every 4 hours.
Or,
℞ Ol. santalis flavæ.....ʒ ij.
Ol. cinnamomi.....ɱ ij.
M. Sig. Take gtt. x-xv. on a piece of sugar t. i. d. (and as much milk as possible, but no spirits).

CRABS.

℞ Tr. delphini.....ʒ i.
Sig. Put on sponge and sponge the parts.

CATHARTIC.

℞ Strych. sulph.....gr. i.
Res. podoph.....gr. x.
Ext. col. co.....gr. xl.
Pulv. capsici.....gr. xx.
M. et ft. pil. xl. sig. One or two when required.

CEREBRAL CONGESTION.

℞ Sodii brom.....ʒ i.
Fl. ext. ergot. (Squibb's)..ʒ iv.
M. Sig. Tablespoonful t. i. d.
Or,
℞ Sodii brom.....ʒ i.
Pep. sacch.,
Lig. carb. pulv.....āā ʒ iij.
Fl. ext. ergot.,
Aquæ.....āā ʒ ij.
M. Sig. ʒ i. t. i. d. for one month.
Then give as a tonic:
℞ Quin. sulph.....gr. iv.
Ac. hydrobrom.....ʒ iv.
M. Sig. ʒ i. t. i. d. after meals.

CHILBLAINS.

℞ Plumb. acet.....ʒ ss.
Aquæ.....O.i.
M. Sig. Apply as a lotion to the affected parts.
Or,
℞ Acid. carbol.....ʒ i.
Tr. iodini.....ʒ ij.
Acid. tannici.....ʒ ij.
Cerat. simp.....ʒ iv.
M. Sig. Ungt.

COLIC.

℞ Sodii bicarb.,
Spt. æther comp. (Hoff.),
Morph. sol.,
Syr. zingiber.....āā ʒ ss.
Aq. camph., ad.....ʒ ij.
M. Sig. ʒ i. repeat till relieved.

COLLYRIA.

℞ Atrop. sulph.....gr. iv.
Aq. dest.....fl. ʒ i.
Or,
℞ Duboisiæ sulph.....gr. ¼.
Aq. dest.....fl. ʒ i.
Or,
℞ Eserinæ.....gr. ij.
Aq. dest.....fl. ʒ i.
Or,
℞ Zinci sulph.....gr. iv.
Aq. dest.....fl. ʒ i.
Or,
℞ Aluminis.....gr. ij.
Aq. dest.....fl. ʒ i.
Or,
℞ Acidi gallici.....ʒ ij.
Glycerinæ.....fl. ʒ i.

CONSTIPATION.

℞ Tr. nuc. vom.,
Tr. bella.,
Tr. physost.....āā ʒ ij.

M. Sig. gtt. xxx. in water morning and evening.

Or,
℞ Ext. physostig.,
Ext. bella.,
Ext. nuc. vom.....āā gr. v.

Ft. pil. x. Sig. one pill at bed-time.

Or,
℞ Aloes soc.,
Sap. Castil.....āā ɔi.
Ext. hyos.....gr. x.
Pulv. ipec.....gr. v.

M. et ft. pil. xx. Sig. take one night and morning.

Or,
℞ Ext. bella.....gr. v.
Ext. nuc. vom.....gr. x.
Ext. col. co.....ʒ i.
Sodii carb. exsiccat.....gr. xl.

M. et ft. pil. xl. Sig. take 2 at bed-time.

CONSTIPATION.

Or (In Children).

℞ Podoph.....gr. i.
Sacch. lactis.....ʒ i.

M. et ft. chart. xx. Sig. take one every morning or night.

Or,
℞ Pulv. rhei.....gr. iij.
Sodii bicarb.....gr. x.
Pulv. zingib.....gr. ij.
M. et ft. chart. i.

Or,
℞ Ext. colyc. co.....gr. ij.
Ext. bella.....gr. ½.
Ext. gent.....gr. i.
Ol. carui.....gtt. ss.
M. et ft. pil. i.

Or,
℞ Resin. podoph.....gr. 1-6.
Pulv. rhei.....gr. ij.
Ext. bella.....gr. ¼
Ol. carui.....gtt. ss.
M. et ft. pil. i.

Or,
℞ Rhei rad.,
Sapon. Castil.....āā ʒ ss.
Ol. anis.....gtt. iv.

M. ft. pil. xx. Take one or two as required.

Or, when obstinate,
℞ Rhei.....ɔij.
Aloes.....ɔi.
Ext. nuc. vom.....gr. iv.

M. et ft. pil. xl. One or two as required.

Or (Chronic in Women).

℞ Ext. bella.....gr. v.
Ext. nuc. vom.....gr. x.
Ext. colocynth. co.....ʒ i.

M. ft. pil. xx. Take one at night. Should griping occur, add sodii bicarb. and make pil. xl. Take 2 at bed-time.

CONSTIPATION.

Or (During Pregnancy).

℞ Ext. aloes.....gr. iv.
Nuc. vom .....gr. i.
Ext. hyos.....gr. iv.
(Or, ext. bella.....gr. i.)
M. et ft. pil. iv. Sig. take one every morning.

Or (In Infants).

℞ Resin. podoph.....gr. i.
Syr. rhei..... ℥ i.
Ol. fœnic.....gtt. i.
M. Sig. gtt. x.-℥ i.

Or (In Women).

℞ Ext. col. co .....gr. iij.
Ext. bella.....gr. ¼.
Saponis.....gr. i.
M. et ft. pil. l. Take one every night.

COUGH.

℞ Morph. sulph.....gr. ij.
Ac hydrocyan. dil..... ℥ ss.
Syr. scillæ,
Syr. tolu..... .....āā ℥ ij.
M. Sig. ℥ i. every 2 hours till relieved.

Or,

℞ Ammon. chlor..... ℥ ij.
Morph. sulph..... .....gr. ij.
Tr. acon. rad.. .....♏ xvi.
Fl. ext. bella.....♏ v.
Fl. ext. glycyrrh.,
Syr. simp.....āā ℥ i.
Aquæ..... ad ℥ iv.
M. Sig. ℥ i. every 2 hours.

Or (Reflex of Pregnancy).

℞ Pot. brom.....gr. xx.
Chloral hydrat.....gr. v.
M. Sig. this amount to be taken at bed-time for 2 or 3 weeks.

Or (When Violent and Troublesome).

℞ Ac. hydrocy. dil.....gtt. xvi.
Syr. prunis virg.....fl. ℥ i.
M. Sig. ℥ i. every 2 or 3 hours.

COUGH.

Or (Troublesome).

℞ Syr. scillæ..... ℥ iss.
Spt. œth r comp. (Hoff).... ℥ i.
Morph. sulph.....gr. i.
Aq. camph.,
Muc. acaciæ..... āā ℥ i.
M. Sig. ℥ i.-iv. t. i. d.

COUGH MIXTURE (Adult).

℞ Syr. senega,
Syr. prun. virg.,
Syr. tolu... ....āā ℥ i.
Tr. opii camph.,
Sp. nit. dulc.....āā ℥ ss.
Ammon. mur..... ℥ ss.
M. Sig. ℥ i. t. i. d.

COUGH SYRUP.

℞ Morph. acet..... . . gr. ¼.
Tr. hyos..... ℥ iss.
Syr. tolut..... ℥ iiss.
Aq .... ℥ ss.
M. Sig. ℥ ij. every three hours.

CHOREA.

℞ Liq. pot. arsen..... ..... ℥ i.

Sig. gtt. ij. t. i. d. and increase gtt. i. every week.

Or,

℞ Elixir phos..... ℥ ij.
Sig. ℥ i. t. i. d.

Or,

℞ Zinci valer..... ..... ℈ ij.
Cinchoniæ sulph..... ℈ i.
M. ft. pil. xx. Sig. one pill t. i. d.

Or,

℞ Zinci brom..... ℥ i.
Syr. simp..... ..... ℥ i.
M. Sig. gtt. x. t. i. d. and increased so soon as the stomach can bear it.

Or,

℞ Strych. sulph.....gr. ij.
Aquæ..... ..... ℥ i.

Sig. gtt. v. t. i. d. to a child from 10 to 15 years old.

CHOLERA INFANTUM.

℞ Tr. opii comp. (Squibb's)... ʒ i.

Sig. gtt. x. every ½ hour in water till relieved.

Or,

℞ Cup. sulph.....gr. i.
Tr. opii deodor.....gtt. viij.
Aq. destil.....ʒ iv.

M. Sig. ʒ i. every 2, 3, or 4 hours till relieved.

Or,

℞ Tr. zingib.,
Tr. menth. pip.....āā ʒ i.
Tr. opii camph.....ʒ ij.
Aq. menth. pip.....ad ʒ ij.

M. Sig. ʒ i. every hour.

Or,

℞ Mist. creta.....ʒ ij.
Tr. catechu,
Lactopeptin,
Bismuth. subnit.....āā ʒ i.
Tr. opii.....gtt. xij.

M. Sig. ʒ i. every hour in water till relieved.

Or (With Vomiting, etc.).

℞ Pepsin (Boudault's).....ʒ ss.
Bismuth. subnit.....ʒ i.
Ingluvin.....ʒ ss.
Tr. opii camph.....gtt. xvi.
Aq. anisæ.....ʒ ij.

M. Sig. ʒ i. every three hours.

Or (In a Child 6 Months Old).

℞ Sodii bicarb.....gr. vj.
Pepsin.....gr. xx.
Ingluvin.....gr. xv.
Pulv. opii.....gr. ss.

M. et ft. chart. vij.

CHOLERA MORBUS.

℞ Plumbi acet.,
Pulv. camph.....āā gr. vi.
Pulv. opii.....gr. i.
Ext. gent.....q.s.

M. ft. pulv. vi. Sig. One every three hours till relieved, and if vomiting very bad give,

℞ Plumb. acet.....gr. viij.
Morphiæ acet.....gr. i.
Aq. camph.....ʒ ij.

M. Sig. ʒ i. every hour.

CHORDEE.

℞ Camphoræ,
Lactucarii.....āā ʒ i.

M. et ft. pil. xxx. Sig. take 1, 2, or more as necessary.

Or,

℞ Vin. colchici.....ɱ xxx.

Sig. to be taken every night.

CHLOROSIS

℞ Tr. ferri perchlor.,
Ac. acet. dil.....āā ɱ xx.
Syrupi.....ʒ ij.
Liq. ammon. acet.....ʒ vij.

M. Sig. 2 to 4 teaspoonfuls every 3 hours.

CROUP.

℞ Hyd. sulph. flav.....gr. xij.
Pulv. sacch. albi.....gr. xxx.

M. et ft.. chart. vi. Sig. give one every ½ hour till vomiting be produced.

CEREBRAL CONGESTION.

(For a Child 6 Years of Age).

℞ Potass. brom.....ʒ iv.
Potass. iodidi.....ʒ ss.
Fl. ext. ergotæ (Squibbs).....ʒ ij.
Syr. rub. idæi,
Aquæ.....āā ʒ j.

M. Sig. ʒ i. every three hours.

DEAFNESS (With Discharge).

℞ Hydrarg. perchlor. .... .gr. v.
Liq. opii.....ʒ ss.
Glycerinæ.....ʒ i.
Aquæ.....ad ʒ iij.

M. Sig. Fill the ear morning and evening with equal parts of hot water and the above preparation.

DEBILITY (With Nervousness.

℞ Pot. brom.....ʒ i.
Ferri sulph. exsicc.....ʒ ss.
Calumbæ,
Zingiberis.....āā ʒ iv.

M. et ft. chart. xxiv. One at bed time.

DELIRIUM TREMENS.

℞ Tr. capsici.....ʒ vi.
Tr. nuc. vom.....ʒ ij.

M. Sig. gtt. xx. in water every 4 hours.

Or,

℞ Morph. sulph. sol.,
Fl. ext. valerian.....āā fl. ʒ i.

M. Sig. ʒ i.-ij. as required.

Or,

℞ Pot. brom. .....ʒ iv.
Chloral hydrat.....ʒ iij.
Aq.....ʒ ij.

M. Sig. ʒ i. every ½ hour till relieved.

Or,

℞ Chloral hydrat.....gr. xxx.
Every ½ hour.

DEPRESSED SPIRITS.

℞ Ac. nit. mur.....ʒ ss.
Sig. gtt. x. t. i. d. in water.

DYSMENORRHŒA.

℞ Pulv. Opii.....gr. ij.
Ol theobro.....q.s.
M. ft. suppos. 1.

DYSMENORRHŒA.

Or (From Anæmia).

℞ Sulph. strych.....gr. i.
Ferri pyrophos.,
*Quin. sulph.....āā ʒ i.
Ac. phos. dil.,
Syr. zingiber.....āā ʒ ij.
M. Sig. ʒ i. t. i. d.

DYSENTERY (With Tenesmus).

℞ Iodoformi,
Pulv. opii.....āā gr. viij.

M. ft. pil. viij. Sig. One every 2 hours unless allayed.

Or (Acute).

℞ Cup. sulph.....gr. ss.
Mag. sulph.....ʒ i.
Ac. sulph. dil.....ʒ i.
Aq.....ʒ iv.

M. Sig. a tablespoonful every four hours.

Or (Asthenic).

℞ Aq. camph.....ʒ iv.
Acidi nitr.....ɱ xxx.
Tr. opii.....ɱ xxx.

M. Sig. A tablespoonful every 2 or 3 hours.

Or (Chronic).

℞ Liq. pot. arsen.....gtt. ij.
Tr. opii.....gr. v.

M. Sig. Take this before each meal.

Or (Chronic).

℞ Cup. sulph.,
Morph. sulph.....āā gr. i.
Quiniæ sulph.....gr. xxiv.

M. et ft. pil. xij. Sig. One t. i. d.

Or (Chronic).

℞ Fl ext. ergot. (Squibb's). ʒ iiiss.
Tr. opii deodor .....ℨ ss.
M. Sig. ʒ i. t. i. d.

* Tr. hydrastis may be substituted.

DYSENTERY.

Or (In a Child 5 Years of Age).

℞ Iodoformi... .....gr. xv.
Mucil. acac..... ʒ i.
Acidi tannici.....gr. xl.

M. Sig. put ʒ i. in some thin boiled starch and *inject* per rectum.

Or (In a Child 5 Years of Age).

℞ Plumb. acet.... ..... gr. xx.
Pulv. opii..... . .. .gr. i.
Ergotin.....gr. iv.
Glycyrrhiz.....q.s.

M. et ft. chart. x. Sig. One every 2 hours.

DYSPEPSIA.

℞ Sodii bicarb.....gr. xx.
Acid carbol.....gtt. ij.
Acac.,
Sacchari albi.. .....āā q.s.
Spts. lavand. co.....ʒ ij.
Aq.....ʒ vi.

M. Sig. ʒ i. two hours after each meal.

Or,

℞ Pulv. pepsin.,
Bismuth. subcarb....āā gr. xx.

M. et ft. chart. x. Sig. take one t. i. d.

Or,

℞ Tr. gent. co.,
Tr. rhei.....āā ʒ ij.

M. Sig. ʒ i. before each meal.

Or (And Vomiting).

℞ Bismuth. subcarb. .....gr. x.
Sodii bicarb.....gr. iij.

M. et ft. chart. i.

Or (Chronic).

℞ Arg. oxidi,
Ext. hyos.....āā gr. v.

M. et ft. pil. x. One t. i. d. before meals.

DYSPEPSIA.

Or (Sour Risings after Meals).

℞ Inf. calumb..... .. .. . ʒ iv.
Liq. potass..... .... ʒ ss.

M. Sig. ʒ ij. t. i. d. half an hour before each meal.

Or (When Hands and Feet Cold).

℞ Asafœtidæ .....gr. iij.-ix.
Sodii bicarb.....gr. ij.
Paradise semen.....gr. i.

M. put the above pill in capsule (one should be taken t. i. d.).

DYSPEPTIC TONIC (After Fevers, etc.).

℞ Ammon. carb.....ɔ iv.
Ferri ammon. cit ..... ʒ i.
Tr. nuc. vom..... ..... ʒ ij.
Tr. quassiæ,
Tr. gent. co.....āā ʒ iv.
Elixir simp..... ... . ʒ iij.
Aq. camph... .....ad O. i.

M. Sig. ʒ ij.-iv. t. i. d. before or after meals.

DIARRHŒA.

℞ Morph. sulph.....gr. ss.
Ol. theobrom.....q. s.
Ft. suppos. No. 1.

Or,

℞ Iodoformi.....gr. v.
Ol. theobrom.....q. s.
Ft. suppos. No. 1.

Or,

℞ Morph. sulph.....gr. ss.
Ext. bella.....gr. iss.
Ol. theobrom.....q. s.
Ft. suppos. 1.

Or,

℞ Morph. sulph.....gr. ss.
Ext. bella .. ..gr. iss.
Ac. tannici.... .....gr. v.
Ol. theobrom.....q. s.
Ft. suppos. 1.

DIARRHŒA.

Or,

℞ Ac. tannici.....gr. v.
Ol. theobrom.....q. s.
Ft. suppos. No. 1.

Or,

℞ Aq. camph.....ʒiv.
Acidi nit.....ɱ xxx.
Tr. opii.....ɱ xx.

M. Sig. A tablespoonful every 2 or 3 hours.

Or,

℞ Pulv. opii.....gr. j.
Plumb. acet.....gr. ij.
M. et ft. pil. i.

Or,

℞ Pulv. opii.....gr. j.
Acid. tann.....gr. iij.
M. et ft. pil. i.

Or,

℞ Spts. lavan. co.....ʒi.
Spts. camphor.....ʒi.
Tr. opii.....ʒss.
Sacch. albi,
Muc. acac.....āāʒiss.
Aq. cinnam.....ad ʒvi.

M. Sig. A tablespoonful every 3 hours.

Or,

℞ Tr. opii.....gtt. xx.
Bismuth. subnit.....ʒij.
Syr. simp.....ʒi.
Mist. cretæ.....ʒiij.

M. Sig. ʒi. every 3 hours.

Or,

℞ Mist. cretæ.....ʒij.
Lactopeptin.....ʒi.
Tr. catechu.....ʒi.

M. Sig. ʒi. every 2 hours.

Or,

℞ Ol. ricini.....ʒi.
Tr. zingib.....gtt. xx.
Tr. opii.....gtt. x.–xx.
Vin. gallici.....ʒi.

M. Sig. Take at once.

DIARRHŒA.

Or,

℞ Bismuth. subnit.....ʒij.
Morph. sulph.....gr. i.

M. et ft. pulv. vi. Sig. ʒi. t. i. d. in milk.

Or,

℞ Ac. sulph. arom.....ɱ xl.
Ext. hæmatoxyli.....ʒi.
Tr. opii camph.....ʒiv.
Syr. zingiberis.....ʒiv.

M. Sig. 2 teaspoonfuls every 3 hours.

Or,

℞ Mist. cretæ.....ʒv.
Tr. catechu,
Tr. opii camph.....āāʒiss.

M. Sig. Dose one teaspoonful every 3 hours.

Or,

℞ Tr. calumb.....ʒxv.
Tr. opii deodor.....ʒi.

M. Sig. ʒi. in wine-glass of water before meals.

Or (Chronic).

℞ Liq. pot. arsen.....gtt. ij.
Tr. opii.....gtt. v.

M. Sig. Take this before each meal.

Or (Chronic).

℞ Cup. sulph.,
Morph. sulph.....āā gr. i.
Quiniæ sulph.....gr. xxiv.

M. et ft. pil. xij. Sig. one t. i. d.

Or (Chronic).

℞ Fl. ext. ergot. (Squibb's).ʒiiiss.
Tr. opii deodor.....ʒss.

M. Sig. ʒi. t. i. d.

Or (In Child 6 to 9 Months, with Green Stools).

℞ Pepsin.....ʒss.
Bismuth. subnit.....ʒi.
Tr. opii.....gtt. xx
Aq. menth. pip.,
Aquæ lauro cerasi.....āāʒi.

M. Sig. ʒi. every 3 hours.

DIARRHŒA.

Or (In Typhoid Fever).

℞ Bismuth. subcarb..gr. xxx.-lx.
Sig. Take this t. i. d. in milk.

DIARRHŒA OF PHTHISIS.

℞ Resin. terebin..... gr. iij.
Argent. nitrat.,
Pulv. opii..... .....āā gr. ¼.

Sig. One pill when needed.

DIARRHŒA (Summer).

℞ Mag. sulph..... ʒ i.
Ac. sulph. dil..... ʒ ij.
Morph. sulph..... .....gr. i.
Aq..... ..... ʒ iv.
M. Sig. ʒ ij. every 3 or 4 hours.

Or (Summer, in Adults).

℞ Mag. sulph.... ..... ʒ vi.
Tr. rhei,
Tr. zingiberis.....āā ʒ iss.
Aq. menth. pip.....ad ʒ vi.
M. Sig. ʒ iv. t. i. d.

Or (Summer, for Children 1 Year and Under).

℞ Mag. sulph..... ʒ i.
Tr. rhei..... ..... ʒ ij.
Syr. zingiber.... ..... ʒ iv.
Aq. menth. pip ... .ad ʒ iss.
M Sig. ʒ i. every 3 hours.

DIPHTHERIA.

℞ Tr. ferri mur. ..... ʒ iss.
Sat. sol. chlor. pot..... ℥ ij.

M. Sig. ʒ i. every 2 hours.

Or,

℞ Tr. ferri persulph..... ʒ ss.

M. Sig. Put ʒ i. in a tumblerful of water and gargle the throat.

Or,

℞ Hydrarg. prot. iodidi,
Pot. bichrom.. ....āā ʒ iv.

M. Sig. Put sufficient in a tumbler of water to make it a deep yellow and take a teaspoonful every hour.

Or,

℞ Pot. chlor..... ʒ ss.
Aq.. ..... ʒ vi.

M. Sig. Tablespoonful t. i. d.

DROPSY.

℞ Vin. colch. sem..... . ʒ ss.
Sol. ammon. acet..... ʒ iiss.
Inf. petroselin.. ..... ℥ v.

M. Sig. A tablespoonful every four hours.

EAR-ACHE.

℞ Chloroformi..... ..... ℥ i.
Vini opii,
Glycerin.... ....āā ℥ ij.
Ol. amygdal.... ..... ℥ iij.

M. Sig. gtt. i. or ij. to be dropped in the ear night and morning.

ECZEMA.

℞ Ung. zinc. ox.,
Ung. hydrarg. nit.,
Ung. plumb. subacet...āā ℥ ss.

M. et ft. ungt. Apply to parts affected.

Or,

℞ Acid. nit. dil..... ℥ ss.
Plumb. acet.....gr. v.
Aq..... ..... ℥ vi.

Sig. Lotion.

Or,

℞ Liq. plumbi subacet. ... ℥ i.
Glycerinæ..... ..... ℥ ss.
Aq. lauro cerasi. .. .... ℥ iiiss.

Sig. Lotion.

Or,

℞ Ac. salycil.,
Pulv. boracis.....āā gr. x.
Aq..... ... .O.i.

M. Sig. Apply to parts affected.

EMETIC.

℞ Ipecac.....gr. xxx.

EMPHYSEMA (With Asthma and Chronic Bronchitis).

℞ Syr. prun. virg.. .. .... ℥ iv.
Spr.œther comp. (Hoff.)..℥ ss.
Pot. iodidi..... ℥ ij.
Tr. bella..... ℥ ij.-iv.

M. Sig. ℥ i. t. i. d. and send the patient to a warm climate.

Or,

℞ Pot. chlor..... ..... ℥ iss.
Tr. bella..... ℥ iss.
Fl. ext. prun. virg.,
Tr. cinchon. co.....āā ℥ ij.

M. Sig. A dessertspoonful 4 times a day.

ENTERITIS.

℞ Pulv. opii..... .....gr. i.
Plumb. acet.....gr. ij.

M. et ft. pulv. i.

ERYSIPELAS.

℞ Vin. ipecac.....fl. ℥ ss.

Sig. gtt. xx. every 2 or 3 hours in water.

Or (Facial).

℞ Quin. sulph..... ..... ℥ ss.
Ext. bella..... ....gr. iij.

M. ft. pil. x. Sig. One every four hours.

EPILEPSY (Idiopathic).

℞ Pot. iodidi,
Pot. brom..... .....āā ℥ *i.*
Ammon. brom..... ℥ ss.
Pot. bicarb..... ℈ ij.
Inf. calumb..... ℥ vi.

M. Sig. ℥ i. before each meal and 3 tablespoonfuls at bed-time with water.

ERYSIPSLAS.

℞ Tr. ferri perchlor.,
Glycerinæ..... ....āā ℥ j.

Sig. ℥ ij. every 2 hours.

FACIAL NEURALGIA.

℞ Ammon. val..... ...gr. v.–x.
Chl. ammon..... ....gr. xxx.
M. et ft. pulv. Sig. Take immediately.

FEVER.

℞ Lig. ammon. acetat..... ʒ ij.
Sp. nit. dulc..
Syr. ipecac..... .....āā ʒ i.
M. Sig. ʒ i. every 2 hours.

Or (Malarial).

℞ Quin. sulph..... ℈ ij.
Fer. sulph. exsic..... ℈ i.
Ac. arseniosi.....gr. i.
M. et ft. pil. xx. Sig. One t. i. d.

FÆCAL ACCUMULATION.

℞ Tr. aloës et myrrh..... ʒ vi.
Tr. nucis vom..... ʒ ij.
M. Sig. gtt. xv.–xxx. t. i. d.

FÆCES (Incontinence of).

℞ Tr. bella..... ..... ʒ v.
Fl. ext. ergotæ,
Aq..... .āā ʒ ij.
M. Sig. ʒ i. t. i. d.

GASTRITIS (Chronic).

℞ Argent. nitratis..... ..gr. v.
Pulv. opii..... ..gr. iiss.
M. et ft. pil. xx. One t. i. d.

GLEET (In Anæmic Persons).

℞ Tr. fer. perchlor..... ..... ʒ vi.
Tr. cantharides..... ..... ʒ ij.
M. Sig. gtt. xv. in water t. i. d.

GONORRHŒA.

℞ Sol. plumb. subacet. (Goulard's)..... .... ʒ ss.-i.
Aq..... ʒ iv.
M. et ft. injectio. Sig. Use once daily.

Or,

℞ Copaibæ,
Pulv. cubebæ..... āā ʒ ij.
Pulv. alum..... ..... ʒ i.
Pulv. opii..... gr. v.
M. Sig. ʒ i.-ij. night and morning.

Or,

℞ Ol. copaibæ,
Ol. cubebæ,
Ol. santal. flav..... āā ʒ i.
Magnesiæ..... ʒ ij.
M. et ft. pil. lx. Sig. Take two pills every 4 hours.

Or,

℞ Vin. colch. sem..... ʒ ss.
Sol. pot. cit..... ..... ʒ viss.
Tr. opii deodor..... ʒ ij.
M. One tablespoonful t. i. d.

Or,

℞ Liq. plumb. subacet. dil. ʒ iv.
Zinci sulph..... .....gr. viij.
M. et ft. injectio.

Or,

℞ Cad. sulph.....gr. i.
Aq..... ʒ iv.
M. et ft. injectio.

Or,

℞ Bals. copaibæ,
Sp. nit. dulc..... āā ʒ i.
Liq. potas..... ʒ ij.
Fl. ext. glycyrrh..... ʒ ss.
Misce et adde:
Ol. gaulther..... ..gtt. xv.
Syr. acac.....ad ʒ vi.
M. Sig. ʒ i. t. i. d.

GONORRHŒA AND GLEET.

℞ Fl. ext. hydrastis..... ʒ i.
Mucil. acac..... ... ʒ iv.
M. et ft. injectio. Sig. Use ʒ ss. as an injection.

Or,

℞ Fl. ext. hydras..... ʒ ss.
Aq..... ʒ ij.
M. et ft. injectio.

GOUT.

℞ Pil. hydrarg.....gr. vi.
Carb. sodii..... .....gr. v.
Ext. col. co..... .. ...gr. vi.
M. et ft. pil. i. Sig. Take at bed time.

Or,

℞ Pulv. ipecac,
Hydrarg, chlor. mite.,
Ext. mellon,
Ext. colch. acet. .....āā gr. i.
Ext. nuc. vom.....gr. ¼.
M. Sig. One pill every 4 hours; if attack be very bad, 1 in every 2 hours. In severe cases, with pain, give gtt. x. Magendie's solution.

Or,

℞ Vin. colch. rad..... ʒ i.
Pot. bicarb.,
Pot. et sod. tart..... āā ʒ iiss.
Aq. menth. pip..... ʒ iv.
M. Sig. ʒ iv. t. i. d.

Or, GOUT.

℞ Pot. carb.,
Pot. nit.....āā ʒ iiss.
Aq .....ʒ viij.

M. Sig. Tablespoonful t. i. d.

Or,

℞ Tr. colch. seminis.....ɱ xx.
Pot. bicarb.....gr. x.
Aq. piment.....ʒ i.

M. Sig. A draught.

Or, GOUT.

℞ Tr. colch. sem.....ɱ xv.
Mag. carb.....gr. vi.
Mag. sulph.....gr. xxx.
Aq. menth. pip. ....ad ʒ i.

M. Sig. A draught.

HAY ASTHMA.

℞ Pot. iodidi..... ℥i.
Liq. pot. arsen..... ℥i.
Aq..... ℥iv.

M. Sig. ℥i. every 4 hours.

Or,

℞ Liq. pot. arsen..... ℥iij.
Tr. bella..... ℥ix.

M. Sig. gtt. xij. after meals and increase in 3 days to xvi. and after 10 days to gtt. xx.

HÆMATEMESIS (From Low Condition).

℞ Ol. terebinth..... ℥iij.
Fl. ext. dig..... ℥i.
Mucil. acac..... ℥ss.
Aq. menth. pip..... ℥i.

M. Sig. ℥i. every 3 hours.

HÆMOPTYSIS.

℞ Plumbi acet.,
Pulv. digit..... āā ℈i.
Pulv. opii..... gr. x.

M. ft. pil. xx. Sig. One every four hours.

Or,

℞ Fl. ext. ergot..... ℥iij.
Fl. ext. ipec.,
Tr. opii deodor..... āā ℥ss

M. Sig. ℥i. every hour.

HEADACHE.

℞ Fl. ext. guaran..... ℥i.
Hoff. anodyne..... ℥ij.
Tr. lavand. co..... ℥vi.

M. Sig. ℥i. every ½ hour till relieved.

Or (Reflex).

℞ Pot. cyanid..... gr. x.-℈i.
Aq. lauro cerasi..... ℥iv.

M. Sig. Poison. A compress moistened with the solution to be applied to the seat of pain.

HEADACHE.

Or (Severe Continuous).

℞ Give hydrarg.chlor.mite. gr. 1-30.
Every 15 minutes.

Or,

℞ Pot. brom..... ℥i.
Pot. iodidi..... ℥ij.
Glycerinæ,
Syr. rub. idæi..... āā ℥i.

M. Sig. ℥i every 3 hours.

Or (Sick).

℞ Ext. gent.,
Pulv. rhei..... āā ℥ss.
Pil. hydrarg..... gr. iv.
Ol. caryophyl..... gtt. iv.

M. et ft. pil. xx.

HEART COMPLICATIONS (In Rheumatism).

℞ Pot. bicarb..... ℥iss.
Pot. acet..... ℥ss.
Pot. nitratis..... ℈i.
Spts. æther. comp..... ℥i.
Ac. citrici..... ℈i.
Aq..... ℥i.

M. Sig. Take half this mixture every hour (making 2 doses).

HEART (Hypertrophy of).

℞ Plumb. acet..... ℥ss.
Pulv. opii..... gr. v.
Confect. ros..... q. s.

M. et ft. pil. xx. One t. i. d.

HEART LESIONS (with Dilatation).

℞ Brom. potas..... ℥iij.
Chloral hydrat..... ℥i.
Syr. rub. idæi,
Aq..... āā ℥ij.

M. Sig. ℥i. every hour till relieved.

HEMIPLEGIA (Due to Syphilis).

℞ Pot. iodidi..... ℥i. (dose).

Give t. i. d. in half a tumbler of infusion of hops, and increase the pot. iodide till ℥i. t. i. d. be reached.

HEMORRHAGE (PULMONARY).

℞ Piumb. acet..... ʒ ss.
M. et ft. pulv. vj. Sig. One every 3 hours.

HEMORRHAGIC DIATHESIS.

℞ Ac. gall..... ʒ ss.
Ac. sulph. dil.,
Tr. opii deodor.....āā ʒ i.
Inf. ros. co..... ʒ iv.
M. Sig. A tablespoonful every 3 hours.

HEMORRHOIDS.

℞ Pulv. hydras.. ..... ʒ iij.
Vaseline..... ʒ i.
Paraffin..... q. s.
M. et ft. ungt. Sig. Apply night and morning.
Or,
℞ Pulv. gall ..... ℈ i.
Pulv. opii.....gr. x.
Ung. plumb. subacet .... ℈ ij.
Ung. simpl..... ʒ i.
Sig. To be used as directed night and morning.
Or,
℞ Pulv. opii..... ..gr. ij.
Ol. theobro.....q. s.
M. ft. suppos. 1.

HEPATIC COLIC.

℞ Sodii phos. ....℈ i.–ʒ i.
Sig. Take this amount before each meal.

HYSTERIA.

℞ Chloral hydrat..... ʒ iss.
Pot. brom..... ʒ iij.
Syr. rub. idæi..... ʒ i.
Aq. aurant. flav..... ʒ iij.
M. Sig ʒ i. t. i. d.

HOOPING COUGH.

℞ Zinci oxidi.. ..... ʒ i.
Ext. bella.....gr. v.
M. et ft. pil. xxi. Sig. One t. i. d.

HOOPING COUGH.

℞ Syr. ipecac..... ..... ʒ iiss.
Syr. scillæ..... ʒ iij.
Mist. assafœt..... ad ʒ ij.
M. Sig. ʒ i.–ij. every 3 hours.
Or,
℞ Pot. brom..... ʒ ij.
Hydrat. chlor. ..... ʒ ss.
Ext. bella..... ʒ i.
Syr. tolu..... ʒ ij.
M. Sig. ʒ i. every half-hour till relieved.
Or,
℞ Camph. monobrom .....gr. v.
Syr. tolu..... .ʒ i.
Mucil. acaciæ .....q. s.
M. Sig. ʒ i. as required.
Or,
℞ Ac. nit. dil ..... ʒ iij.
Tr. cardamom. co.... .... ʒ i.
Syr. simp .... ʒ iij.
Syr. cinnamon..... ʒ vi.
M. Sig. ʒ ij. every three hours and let them drink freely of
℞ Ac. phos. dil.. ..... ʒ i.
Aq ..... O.i.
Or (IF SEVERE).
℞ Fl. ext. hyos ..... ʒ ss.
Aq. aurant..... ʒ iv.
M. Sig. ʒ i.–iv. every three hours.

HORRORS.

℞ Pot. brom.,
Aq.....āā ʒ i.
M. Sig. ʒ ij. every 4 hours.

HYDROPHOBIA.

℞ Inf. xanthis spinosi.. ...gr. ss.
M. Sig. Take this amount t. i. d. for 6 weeks (for adults); for children under 12 years of age, give ½ this dose.

INFLAMMATION OF BLADDER.

℞ Tr. bella..... ʒ i.
Fl. ext. buchu.. ..... ʒ i.
Liq. pot. arsen..... ʒ ss.
Tr. opii camph..... ʒ vi.
Syr. simp..... ʒ iss.
Aq..... ad ʒ vi.
M. Sig. ʒ i. every 3 hours (tr. opii camph. should not be included when blood exists in the urine).

Or,

℞ Pot. brom... ..... ʒ iij.
Chloral hydrat..... ʒ iss.
Syr. rub. idæi,
Aq..... āā ʒ i.
M. Sig. ʒ i. every half-hour till relieved (and apply hot fomentations).

INFLAMMATION OF BOWELS.

℞ Pulv. opii..... gr. i.
Plumb. acet..... gr. ij.
M. ft. pulv. i.

INDIGESTION.

℞ Cinchon. sulph..... gr. ij.
Ext. glycyrrh..... ..... gr. iij.
Sacchari albi ... . gr. v.
M. et ft. chart. i.

Or,

℞ Sodii bicarb.,
Pulv. zingib.,
Pulv. calumb..... āā gr. iiss.
M. et ft. chart. i.

Or,

℞ Bismuth. subnit. ..... gr. xv.
Ft. chart. i.

IRITIS.

℞ Morph. sulph..... gr. iv.
Zinci sulph..... gr. iij.
Atrop. sulph..... ..... gr. ij.
Aq. destil..... ..... ʒ i.
M. Sig. lotio.

ITCHING (Obstinate).

℞ Ac. hydrochlor.... ... gtt. xx.
Aq..... ʒ iv.
M. Sig. Apply to parts.

IMPOTENCE.

℞ Ferri arsen..... ....gr. v.
Ergotine (aq. ext.) ... . ʒ ss.
M. ft. pil. xxx. Sig. Take one night and morning.

INCONTINENCE OF URINE.

℞ Acid benzoic..... . ʒ ij.
Aq. cinnam..... ʒ vi.
M. Sig. A tablespoonful t. i. d.

INCONTINENCE OF URINE AND FÆCES.

℞ Tr. bella..... ʒ v.
Fl. ext. ergot.,
Aq ..... āā ʒ ij.
M. Sig. ʒ i. t. i. d.

INSOMNIA (From Worry).

℞ Pot. brom..... ..... ʒ vi.
Aq..... ... ʒ iij.
M. Sig. ʒ i. before each meal and take ʒ ij. at bed-time.

INFLUENZA.

℞ Vin. ipecac..... ℥ xvi.
Tr. opii..... ℥ xiij.
Spt. æther. nit..... . ʒ i.
M. To be taken, in water, at bed-time.

Or (Of Old People).

℞ Ammon. carb..... ʒ i.
Muc. acac..... fl. ʒ iv.
Aq. menth. pip..... . ʒ ij.
M. Sig. ʒ i.-ij. every hour.

Or (After Loosening of Cough).

℞ Syr. scillæ..... ʒ iij.
Tr. opii camph..... ʒ i.
M. Sig. ʒ i. t. i. d. or ʒ ij. at night.

ITCH.

℞ Sulph. iodidi..... ..... ℈ i.
Adipis..... ʒ i.
M. Apply to the part.

Or,

℞ Sulphuris..... .... ʒ i.
Adipis..... ʒ ij.
M. et ft. ungt.

JAUNDICE.

℞ Pot. bitart..... ℥ i.
Ext. taraxx..... ʒ ss.
Decoct. tarax..... ℥ viij.

M. Sig. Half a wine-glassful t. i. d.

Or,

℞ Sodii phos.....dose ʒ i.–℥ i.
For children..... " gr. x.–ɔ i.

Or (CATARRHAL).

℞ Spt. œther. comp. (Hoff.).. ʒ i.
Sodii bicarb.....gr. x.
Sig. Take this dose t. i. d.

JOINTS (SWOLLEN).

℞ Pot. iodidi..... ʒ i.
Glycerin... ..... ℥ i.
Tr. sap. camph..... ℥ iij.
Ol. limonis.....gtt. iv.
M. et ft. liniment.

LABOR (Weariness or Over-exertion in).

℞ Hydrat chloral.....gtt. viij.
Liq. morph. sulph. (Magendie).....gtt. iij.
Aq. camph..... ʒ i.
M. Sig. ʒ i. every hour till sleep is produced.

LARYNGITIS.

℞ Zinci sulph.....gr. viij.
Acidi tannic.....gr. xx.
Aq ..... ʒ iv.
M. et ft. gargle.

LAXATIVE.

℞ Mag. carb.,
Mag. sulph.,
Sulphuris loti.,
Sacch. alb.....āā ʒ iv.
Pulv. anis..... ʒ ij.
M. et ft. chart. i.

Or (Cooling).

℞ Pulv. ipecac..... gr. 1-6.
Pulv. rhei.....gr. ij.
Sodii bicarb.,
Pulv. cubeb.....āā gr. iv.
M. et ft. chart. i.

LEUCORRHŒA.

℞ Alum..... ..... ʒ ij.
Aq..... ʒ viij.
M. Sig. Inject into vagina bis die.

LICE (To Destroy).

℞ Hyd. chlor. cor..... ɔ i.
Aq..... ʒ iv.
M. Sig. Use as a wash.

LOTION.

R Liq. plumb., subacetat..... ʒ j.
Aquæ .....Oj.
M. Sig. External application.

LINIMENT.

℞ Veratriæ... .. ..gr. iv.
Chloroformi..... ʒ i.
Tr, acon. (Fleming's)..... ʒ ij.
Tr. capsici..... ʒ ss.
Tr. menth. pip..... ʒ iss.
Ol. menth. pip..... ʒ ss.
M. et ft. lotio. Sig. Poison.

Or,

℞ Fl. ext. acon. rad.,
Chloroformi... .....āā ʒ ℓ.
Spt. camph..... ʒ ss.
Lin. sap..... ....q. s. ʒ ij.
M.

LIVER (Enlarged).

℞ Quiniæ sulph. ....gr. x.
Pulv. capsici... .....gr. i.
Pulv. camph..... gr. ss.
M. Sig. Take this dose t. i. d. (in capsule).

Or,

℞ Granules of arsenious acid.....gr. 1-16.
T. i. d.

Or,

℞ Warburg's tinct..... ʒ ij.
T. i. d. for an adult.
Warburg's tinct..... ʒ i.
T. i. d. for children.

LOTION (Red).

℞ Zinc suph.....gr. ij.
Tr. lavand co..... ɱxv.
Aq..... ʒ i.

MALARIAL FEVER.

First give an emetic, then:

℞ Quin. sulph.....℈ ij.
Ferri sulph. exsic.....℈ i.
Ac. arsen.....gr. i.

M. et ft. pil. xx. Sig. One t. i. d.

Or (With Cerebral Symptoms).

℞ Quin. sulph.....gr. v.–x.
Pot. brom.....ʒ ij.–iv.
Syr. rub. idæi,
Aq.....āā ʒ i.

M. Sig. ʒ i. every hour.

Or (In Child 18 Months of Age).

℞ Quin. sulph.....gr. xij.
Lactopeptin.....gr. xv.
Hydrarg. submur.....gr. i.

M. et ft. chart. vi. Sig. One t. i. d.

Or (In Pregnancy).

℞ Ol. eucalypt. glob.....gtt. lx.

M. (Put into 12 capsules). Sig. One every 3 hours.

Or (With Vomiting).

℞ Quin. sulph.....gr. xx.
Bismuth. subnit.....ʒ iss.
(Vel oxalatis cerii .... gr. viij.)
Morph. sulph.....gr. ss.

M. et ft. chart. iv. Sig. One t. i. d.

MARASMUS.

℞ Pepsin..... gr. xxiv.

M. et ft. pulv. viij. Sig. One t. i. d.

MASTURBATION.

℞ Pot. brom.....ʒ iv.
Tr. cinchonæ,
Aq .....āā ʒ iss.

M. Sig. ʒ i. t. i. d.

MENORRHAGIA.

℞ Ferri arsen .....gr. v.
Ergotinæ (aq. ext.).....ʒ ss.

M. et ft. pil. xxx. Sig. One night and morning.

MENSES (Stoppage of).

℞ Pot. brom.....ʒ iv.
Aq .....ʒ ij.

M. Sig. ʒ i t. i. d.

MERCURIAL POISONING.

℞ Pot. iodidi.....gr. x.

Give t. i. d.

MILK FEVER.

℞ Tr. acon. rad.....gtt. xx.
Ant. et pot. tart.....gr. ij.
Sp. etheris nit.,
Syr. simp .....āā ʒ i.
Aq. aurantii flor .....ʒ ij.

Sig. ʒ i. in a wineglassful of water (with sugar) every 2 hours.

MITRAL REGURITATION.

℞ Ferri redact.,
Quin. sulph.....āā ℈ i.
Pulv. dig.,
Pulv. scillæ .....āā gr. x.

M. et ft. pil. xx. Sig. One t. i. d.

MOUTH (Ulceration of).

℞ Pot. chlor.....ʒ ss.
Aq.....ʒ vi.

M. Sig. Tablespoonful t. i. d.

MOUTH WASH.

℞ Sodii bor.....ʒ ij.
Pulv. myrrh.....ʒ i.
Aq.....ʒ vi.

M. ft. wash or gargle.

NERVOUSNESS (With Debility).

℞ Pot. brom..... ʒ i.
Ferri sulph. exsic..... ʒ ss.
Calumbæ,
Pulv. zingiberis, ...āā ʒ iv.

M. et ft. chart. i.

NEURALGIA.

℞ Aconitin..... .... gr. i.
Strychniæ ....gr. iij.
Hydrarg. iod.....gr. vi.
Adipis..... ʒ ij.
Vermilion. .....q. s.

M. et ft. ungt. Rub the affected part well with a very small piece of the ointment.

Or,

℞ Tr. acon. rad.,
Chloroformi.....āā ʒ ss.
Lin. sap..... ʒ i.

M. Sig. Apply to the seat of pain.

Or,

℞ Aconitiæ.....gr. iv.
Veratriæ..... ..gr. xv.
Glycerin ..... ʒ ij.
Cerati ..... ʒ vi.

M. Sig. To be rubbed over the part. Take care there is no abrasion.

Or, NEURALGIA.

℞ Ext. hyos.,
Ext. lupuli.....āā gr. x.
Morph. sulph.....gr. iss.

M. ft pil. vi. Sig. One night and morning.

Or (Facial).

℞ Ammon. val. .....gr. v.–x.
Chl. ammon .. ..gr. xxx.

Sig. Take immediately.

Or (Sacral).

℞ Ol. crotonis..... ʒ i.
Ol. amygd. dulc ... . ʒ ix.

M. et ft. embrocatio.

NOCTURNAL INCONTINCE OF URINE IN CHILDREN.

℞ Strych..... .....gr. i.
Pulv. canthar..... .gr. ij.
Morph. sulph.... ..... .gr. iss.
Ferri pulv.... ..... ℈ i.

M. ft. pil. xl. Sig. One t. i. d. to a child 10 years of age.

OINTMENT (For Sores).

℞ Zinci oxidi.....gr. xx.
Cerati simp.,
Vaseline.....āā ʒ ss.

M. et ft. ungt.

PAINS (AFTER LABOR).

℞ Sol. morph. sulph (U. S. P.). ʒ j.
M. Sig. ʒ i. every 2 hours till relieved.

PERIOSTITIS.

Give pot. iodidi gr. v. to commence with, and increase gradually to gr. xxx.

PERITONITIS (LOCAL).

℞ Morphiæ.....gr. i.
Camphoræ.....gr. xij.
Pulv. sacch. alb. ....gr. xxiv.
M. et ft. chart. vi. Sig. One every 3 hours till relieved, and apply flaxseed poultice.

PERITONEUM, INFLAMMATION OF, AND OTHER INFAMMATORY CONDITIONS.

℞ Tr. acon. rad..... ..... ʒ i.
Adipis..... ʒ iij.
Chloroformi..... ʒ i.
Morph. mur.....gr. vi.
M. ft. linctus. Sig. Apply on cotton wadding to a *small* surface only.

PLEURISY (ACUTE).

℞ Hydrarg. chlor. mit. gr. vi.
Pulv, opii.. . .. .gr. iij.-vi.
Antimon. tart.. .... gr. iss.
M. et ft. pulv. xij. Sig. Take one every 3 hours in water.

Or (SUBACUTE).

℞ Pot. acet.,
Inf. digital.....āā ʒ ij.-iv.
Sig. This amount each day.

Or,

℞ Pulv. dig.,
Pulv. scillæ mer.,
Hyd chlor. mit.....āā gr. x.
M. et ft. pil. x. Sig. One pill t. i. d.

PLEURISY.

Or (PAIN).

℞ Tr. acon. rad..... ..... ʒ i.
Adipis..... ʒ iij.
Chloroformi..... ʒ i.
Morph. mur.....gr. vi.
M. ft. linctus. Sig. Apply on cotton wadding to a *small* surface only.

PNEUMONIA (CASEOUS).

℞ Inf. dig..... ʒ iv.
Plumbi acet..... ..... ℈ i.
Tr. opii.... .... ʒ i.
Sig. A tablespoonful bis die.

Or (TYPHOID).

℞ Ammon. carb..... .... ʒ i.
Muc. acac.....fl. ʒ iv.
Aq. menth. pip..... ʒ ij.
M. Sig. ʒ i.-ij. every hour.

PREGNANCY (VOMITING OF).

℞ Bismuth. subnit.. ... ʒ iij.
Ac. carbol.....gr. ij.-iv.
Muc. acac ... . ʒ i.
Aq. menth. pip.... .. ʒ iij.
M. Sig. ʒ ij. t. i. d.

Or (VOMITING OF).

℞ Cerii oxalat.,
Pulv. ipecac..... ....āā gr. i.
Creasoti.....gtt. ij.
M. et ft. pulv. Sig. To be taken every hour.

Or (SICK STOMACH OF).

℞ Rad. calumbo,
Rad. zingiber.....āā ʒ ss.
Fol. sennæ..... ʒ i.
Aq. bullient.....O.i.
M. Infus. Sig. A wineglassful before each meal.

PILES OR HEMORRHOIDS.

℞ Pulv. opii..... .gr. ij.
Ol. theobrom.....q. s.
M. et ft. suppos. 1.

PHTHISIS.

℞ Ol. morrh..... ʒ i.
Tr. iodinii co..... ..... ɱ viij.

M. Sig. ʒ i.–iv. every 3 hours.

Or,

℞ Mist. ol. morrh.... ..... ʒ i.
Ætheris..... ɱ xvi.

Or,

℞ Ac. carbol.... ..... ʒ iij.
Ol. gaulther. .. ..gtt. xx.
Glycerin..... ..... ʒ i.
Spts. alcohol..... ʒ ss.
Aq.....ad ʒ viij.

M. Sig. Put one tablespoonful in O.ss. water and inhale the vapor.

Or,

℞ Sulph. morph.... .. ..gr. i.
Syr. bals. tolu,
Syr. prun. Virg ..... āā ʒ i.

M. Sig. ʒ i. t. i. d. If there be night-sweats add:

Morphine.. ....gr. 1-60.

Or,

℞ Sulph. morph.... .....gr. i.
Vini antimon..... ʒ ij.
Syr. tolu..... ʒ iss.
Aq..... ʒ iij.

M. Sig. ʒ i. every 3 hours.

Or (Aphthæ of).

℞ Quin. sulph..... .....gr. i.
Olei piperis nig..... .. .gtt. i.
Aq..... ..... ʒ i.

Sig. Apply with a brush or rinse out the mouth.

Or (Fibroid).

℞ Fl. ext. ergotæ,
Tr. hyos..... ....āā ʒ iv.

M. Sig. gtt. xx. t. i. d.

Also,

℞ Protagon..... ..... ʒ i.
Glycerin..... ..... ʒ ij.

M. Sig. ʒ ss. after each meal.

PHTHISIS.

Or (Troublesome Cough).

℞ Syr. prunis Virg.,
Syr. lactucarii.....āā ʒ ij.

M. Sig. One or two dessertspoonfuls at night or ʒ ij. during the day.

Or (When Temperature Rises Above 100°).

℞ Fl. ext. gelsemii,
Tr. ver. vir.....āā ʒ i.
Tr. acon. rad..... ʒ ij.
Tr. digitalis..... ..... ʒ iij.

M. Sig. gtt. vij. t. i. d. If irregularity of pulse exist, omit the digitalis.

PROSTRATION (General).

℞ Ammon. carb..... ʒ i.
Muc. acac.....fl. ʒ iv.
Aq. menth. pip..... ... ʒ ij.

M. Sig. ʒ i.–ij. every hour.

PUERPERAL CONVULSIONS.

℞ Chloral hydrat..... ʒ iij.
Syr. rub. idæi..... ʒ i.

M. Sig. ʒ i. every 20 minutes.

PURGE.

℞ Pil. hydrarg.....gr. vi.
Sodii bicarb.... ... .gr. v.
Ext. coloc. co..... .....gr. vi.

M. Sig. To be taken at bed-time.

PRURITUS.

℞ Ac. carbol..... ʒ i.–ij.
Glycerin..... ..... ʒ i.
Aq.....ad ʒ viij.

M. Sig. Use as a lotion.

PRURITUS ANI.

℞ Pot. brom.,
Chl. hydrat.....āā gr. x.–xv.
Aq.....ad ʒ i.

M. Sig. To be taken at bed-time.

Also, PURITIS ANI.

℞ Sodii bibor..... ʒ ij.
Morph. mur.....gr. xvi.
Ac. hydrocyan. dil..... ʒ ss.
Glycerin..... ʒ ij.
Aq.....ad ʒ viij.

M. Sig. To be applied to the affected part.

PROSTITIS WITH CYSTITIS.

℞ Tr. hyoscyam..... ʒ i.
Potass. bicarb..... ʒ iij.
Aquæ..... ʒ iij.
Mi Sig. ʒ iv. t. i. d.

Or, PRURITIS ANI.

℞ Chloroformi..... ʒ ij.
Glycerin..... ʒ ss.
Cerat. simp..... ʒ iss.

M. et ft. ungt. Apply locally.

RHEUMATISM (Acute).

℞ Ac. salicyl.....ʒ iij.
Sodii bicarb.....ʒ ij.
Glycerinæ,
Aq.....āā ʒ ij.

M. Sig. A tablespoonful every two hours.

Or (Chronic).

℞ Tr. guaiac.....ʒ ij.

Sig. ʒ i. t. i. d. in water.

RHEUMATIC PAINS.

℞ Lin. acon.,
Lin. bella.....āā ʒ ij.
Glycerinæ.....ad ʒ ij.

Sig. Apply locally over seat of pain.

RUM STOMACH.

℞ Tr. nuc. vom.....gr. v.–xv.
Tr. gent. co.,
Tr. calumbæ co...āā ʒ i.

Sig. Take before meals.

SMALL-POX (To Prevent Pitting).

℞ Pulv. carb. lig..... ʒ ss.
Ol. picis,
Cerati simp.....āā ʒ ij.
Syracena purpura..... ʒ ss.

M. et ft. ungt. Sig. Rub over all parts that are exposed.

SPRAIN.

℞ Liq. plumb. subacet. dil. ʒ iss.
Morph. acet..... ʒ i.
Aq..... ʒ viij.

M. et ft. lotio. Sig. For external application.

Or,

℞ Lin. sapon..... ʒ iv.
Aquæ ammon..... ʒ ss.

M. et ft. embrocatio.

Or,

℞ Chloroformi.....fl. ʒ ij.
Tr. acon. rad.,
Aq. ammon.....āā fl. ʒ ss.
Ol. olivæ.....fl. ʒ v.

M. Sig. Liniment for external application.

Or (Of Ligaments).

First place the limb for five minutes in a hot salt and water bath, then apply:

℞ Chloroformi,
Tr. aconiti,
Tr. opii.....āā ʒ iij.
Lin. sap..... ʒ iv.

M. et ft. lotio. Sig. To be used as directed.

SEDATIVE.

℞ Pot. brom.,
Sp. ammon. arom.....āā ʒ ij.
Syr. scillæ..... ʒ i.
Aq..... ʒ iij.

M.

SPERMATORRHŒA.

℞ Ferri arseniat..... ....gr. v.
Ergotinæ (aq. ext.) ..... ʒ ss.

M. et ft. pil. xxx. Sig. One night and morning.

Or (With Impotence).

℞ Tr. canthar.....gtt. vi.
Tr. ferri perchlor..gtt. xv.-xx.
Sig. t. i. d. in water.

SPLEEN (Enlarged).

(See liver enlarged.)

SWELLING OF JOINTS.

℞ Ol. sassafras..... ʒ ij.
Aq. ammon..... ʒ iv.
Lin. sap. camph..... ʒ iij.

M. ft. liniment (for external application).

SICK HEADACHE (Due to Gastritis).

℞ Pulv. calumbæ,
Bismuth. subnit..... āā gr. x.

M. Sig. Take this t. i. d. after meals.

And,

℞ Inf. cinchon..... ʒ ss.-ij.
Take this before meals t. i. d.

SICKNESS OF STOMACH.

℞ Bismuth. subcarb.....gr. xxx.
Ingluvin..... .....gr. xx.

M. et ft. pulv. v. (for adult). Sig. 1 t. i. d.

SKIN DISEASES.

℞ Liq. pot. arsen..... ʒ ss.
Sig. gtt. v. t. i. d.

Or (Due to Syphilitic Origin).

℞ Liq. arsen. et hyd. iodidi.. ʒ ss.
Give ♏ ij.-v. t. i. d.

SKIN ERUPTIONS.

℞ Ungt. hyd. nit. mit..... ʒ ss.
Sig. For external application.

SYPHILIS.

℞ Hyd. protiodidi..... ..gr. xij.
Rosæ confect..... ..ʒi.

M. et ft. pil. xij. Sig. Take one bis die.

Or (Constitutional).

℞ Pot. iodidi..... ʒss.
Aq. cinnamomi..... ʒi.

M. Sig. ʒi. t. i. d.

Or,

℞ Pot. iodidi..... .....gr. xx.
Hyd. chlor. cor.....gr. 1-6.
Aq. cinnam..... ʒi.

M. Sig. ʒij.-iv. every 3 or 4 hours.

Or,

℞ Hyd. chlor. cor.....gr. ½.
Pot. iodidi.....gr. lxxx.
Aq. menth. pip..... ʒi.

M. Sig. ʒi.-ij. every 3 or 4 hours.

Or (Secondary).

℞ Liq. arsen. et hydrarg iodid. ʒiss.
Sig. gtt. iij.–v. t. i. d.

Or (2d and 3d Stages).

℞ Hydr. bichlor.....gr. ij.
Pot. iodidi..... ...ʒss.
Pot. chlor..... ʒi.
Tr. gent. co.,
Aq.....āāʒiv.

M. Sig. ʒi. t. i. d. after meals.

Also,

℞ Pil. hyd. prot.....gr. ss.

In the middle of the day.

Or (Infantile).

℞ Hydrarg. bichlor.. .....gr. i.
Pot. iodidi..... ʒiv.
Syr. aurant.,
Aq.....āāʒij.

M. Sig. gtt. v. for child about one month old and increase to xv.–xx. if the disease does not yield.

SCROFULA.

℞ Iodini..... ....ʒvi.
Pot. iodidi. .....(troy)ʒiss.
Aq. destil.... ..... O.i.

M. Sig. gtt. v.–vi. bis die, in water.

Or,

℞ Syr. fer. iodidi.....fl. ʒss.
Sig. gtt. x.–xx. in water t. i. d.

SORE NIPPLES.

℞ Plumbi nit.....gr. x.–xx.
Glycerin..... ʒi.

Sig. Rub on the affected part.

Or,

℞ Aquæ rosæ..... ..... ʒiiiss.
Liq. plumb. subacet. dil. ʒss.
Ext. opii aq..... ʒi.

Sig. Lotion. First apply a bread and milk poultice to the parts, and after removal, apply a piece of linen of two thicknesses, after being wetted with the lotion.

SORES (Bad).

℞ Ac. carb.?.....gr. ij.
Aq.....O.i.

M. et ft. lotio.

Or (Bed).

℞ Alum..... ..... ʒss.
Tr. camph..... ʒij.

Add the white of four eggs. M. Sig. Apply to the parts affected.

STOMACHIC.

℞ Ac. nitro-mur. dil.. .. ....ɱlxl.
Inf. gent. co..... ..... ʒi.

M. Sig. ʒij. every 3 or 4 hours.

Or,

℞ Liq. pot. arsen..... ʒss.
Tr. quassiæ..... ʒij.
Syrupi..... ʒvss.

M. Sig. ʒi. (diluted) immediately after meals.

SUNSTROKE.

℞ Hydr. bichlor.....gr. i.
Pot. iodidi.....gr. viij.
Aq.....℥iiss.
M. Sig. ʒi. t. i. d.

STRYCHNIA POISONING.

℞ Nicotinæ.....gr. ss.
Aq. dest.....ʒij.
M. Sig. Give ♏x. as occasion requires.

TAPE WORM.

℞ Insert a tube down the œsophagus to the stomach and pour down it from 200 to 400 centigrammes of a very concentrated decoction of pomegranate root, and let the patient fast for 24 hours.

Or,

Let him drink soda water ad lib.

TONIC.

℞ Sulph. strych.....gr. i.
Ferri pyrophos.,
Quin. sulph.....āā ʒ i.
Ac. phos. dil.,
Syr. zingiber.....āā ʒ ij.

M. Sig. ʒ i. t. i. d.

Or,

℞ Ferri sulph.....gr. ij.
Mag. sulph.....ʒ iss.
Ac. sulph. dil.....ɱxv.
Inf. quassiæ.....ʒ i.

M. Sig. Tablespoonful every three hours.

Or,

℞ Vin. ferri.....ʒ iij.
Liq. pot. arsen.....ʒ ss.
Syrupi.....ʒ iss.
Aq.....ʒ iij.

M. Sig. A tablespoonful diluted, after meals.

Or,

℞ Ferri et quin. cit.....ʒ ss.
Syr. simp.....ʒ ij.
Aq.....ʒ vi.

M. Sig. ʒ i. t. i. d.

Or,

℞ Quin. sulph.....gr. x.
Ac. sulph. dil.....q.s.
Glycerin.....ʒ ij.
Aq.....ad ʒ i.

M. Sig. ʒ i. t. i. d.

Or

TONIC.

℞ Cinch. sulph.....ʒ i.
Tr. ferri chlor.....ʒ iv.
Tr. quas.....ʒ i.
Aq.....ad ʒ iv.

M. Sig. ʒ i. t. i. d.

Or,

℞ Liq. pot. arsen.....ʒ ij.
Vini ferri.....ad ʒ iv.

M. Sig. ʒ i. t. i. d. in a wineglass of water after meals.

Or (CHALYBEATE).

℞ Ferri phos.....ʒ i.

Divide in chart. xij. Sig. Take one, in water t. i. d.

TONIC AND ALTERATIVE.

℞ Ol. phosphor.....ɱxvi.
Ol. gaulther.....ɱviij.
Muc. acac.....ʒ i.

M. Sig. ʒ i.–ij. every 3 hours.

Or,

℞ Pot. chlor.....ʒ ss.
Tr. ferri chlor.....ʒ i.
Aq.....ʒ vij.

M. Sig. ʒ i. every 3 hours.

TONIC AND SOOTHING.

℞ Ext. nuc. vom.....gr. iv.
Ext. hyos.....gr. xv.
Pulv. aloes,
Quin. sulph.,
Ferri sulph.....āā ℈ i.

M. et ft. pil. xx. Sig. Take one twice a day.

TONSILLITIS.

℞ Vin. ipecac.....fl. ʒ ss.

Sig. gtt. xx. every 2 or 3 hours in water.

Or,

℞ Quin. sulph.....gr. x.–xx.

Give twice a day.

Or

TONSILLITIS.

℞ Tr. guaiac..... ʒ ij.
Yolk of egg.

M. Sig. ʒ ss. every 4 hours.

Or,

℞ Pot. chlor.,
Alum.....āā ʒ ij.
Ext. bella..... ʒ iv.
Glycerin.,
Aq. rosæ.....āā ʒ ij.

M. et ft. garg. Sig. Use four times daily.

TORPOR OF INTESTINES.

℞ Ext. physos.,
Resin. podoph.....āā gr. iij.

M. et ft. pil. vi. Sig. Take one at bed-time.

Or,

℞ Tr. physost.,
Tr. nuc. vom.,
Tr. bella.....āā ʒ ij.

M. Sig. gtt. xxx. in water night and morning.

THROAT (Ulceration of).

℞ Pot. chlor..... ʒ ss.
Aq..... ʒ vi.

M. Sig. Tablespoonful t. i. d.

TYPHLITIS.

℞ Pulv. opii,
Pulv. ipec.....āā gr. vi.

M. et ft. pil. xij. One every three hours.

TYPHOID PNEUMONIA.

℞ Ammon. carb..... ʒ i.
Muc. acac.....fl. ʒ iv.
Aq. menth. pip..... ʒ ij.

M. Sig. ʒ i.-ij. every hour.

URINE (Incontinence of).

℞ Tr. bella ..... ʒ v.
Fl. ext. ergot.,
Aq.....āā ʒ ij.

M. Sig. ʒ i. t. i. d.

URETHRITIS.

℞ Copaibæ..... ʒ i.
Pulv. acac..... ʒ iij
Mix well and add
Liquor potass..... ʒ iiss.
Glycerinæ,
Spts. nit. dulc.....āā ʒ iss.
Sig. ʒ iv. t. i. d.

VAGINITIS.

℞ Ac. carbol..... ʒ ss.
Glycerinæ.....
Cerati simp.....āā ʒ i.

M. et ft. ungt.

Or,

℞ Ac. carbol..... ʒ i.
Ung. zinci ox..... ʒ i.

Or,

℞ Ac. tannici.....gr. xxx.
Adipis..... ..... ʒ i.

Or,

℞ Camph.,
Chloral hydrat.....āā ʒ ij.
Aq. rosæ..... ..... ʒ iv.

M. et ft. lotio.

VARICOSE VEINS.

℞ Iodini .....gr. i.
Pot. iodidi..... ʒ iiss.
Tr. aurant. co.,
Tr. cinch. co.... .....āā ʒ i.
Aq ....ad ʒ ij.

M. Sig. ʒ i. t. i. d.

VOMITING.

℞ Sodii bicarb..... .. .... ℈ iv.
Spt. œther comp. (Hoff.).. ʒ i.
Morph. Sulph..... ʒ iiss.
Aq. cinnamom.....ad ʒ iv.

M. Sig. Take one or two teaspoonfuls at once.

Or,

℞ Oxalat. cerii.....gr. xxxv.
Lactopeptin.... .....gr. xxx.

M. et ft. chart. vi. Sig. One every 2 hours till relieved.

VOMITING.

Or (Of Pregnancy).

℞ Ingluvin.....gr. xx.
Oxalat. cerii.....gr. xxv.

M. et ft. chart. vi. Sig. One every 4 hours.

Or (Of Pregnancy).

℞ Cup. sulph.....gr. ij.
Aq. destil..... ..... ʒ ss.
M. Sig. gtt. vi. t. i. d.

Or (Of Pregnancy).

℞ Bismuth. subnit..... ʒ iij.
Ac. carbol.....gr. ij.–iv.
Muc. acac..... .. ʒ i.
Aq. menth. pip..... ʒ iij.

M. Sig. ʒ ij. t. i. d.

Or (Of Pregnancy).

℞ Cerii oxalat..... .....gr. i.
Ipecac..... ....gr. i.
Creasoti.....gtt. ij.

Sig. To be taken every hour.

Or (Of Teething).

℞ Bismuth. subnit..... ʒ iij.
Ac. carbol.....gr. ij.–iv.
Muc. acac.... ..... ʒ i.
Aq. menth. pip ..... ʒ iij.

M. Sig. ʒ i. (small, for a child) t. i. d.

Or (Of Teething)

℞ Bismuth. subcarb..... ʒ ij.
Morph. sulph.... .....gr. ss.

M. et ft. chart. vi. Sig. One t. i. d. in milk.

WAKEFULNESS (From Worry).

℞ Pot. brom..... ℥ vi.
Aq..... ℥ iij.

M. Sig. ℥ i. before each meal, and take ℥ ij. at bed-time.

WORMS.

℞ Santonini.....gr. i.-iij.
Sacchari albi .....gr. ij.
Ft. chart. i.

Or (Round and Ascaris Vermicularis).

℞ Fl. ext. spigeliæ,
Fl. ext. sennæ ..... āā ℥ ss.

M. Sig. ℥ i. to a child 3 or 5 years of age.

Or,

℞ Fl. ext. spigeliæ,
Fl. ext. sennæ..... āā ℥ ss.
Santonini.....gr. viij.

M. Sig. ℥ i. to a child 5 years of age.

Or

℞ Hyd. chlor. mit.....gr. xxx.
Santonin .....gr. xij.

M. et ft. pulv. vi. Sig. One t. i. d.

Or,

℞ Pulv. santon.....gr. x.
Hyd. chlor. mit.....gr. iij.
Res. jalapæ.....gr. i.
Sacch. lactis.....gr. xx.

M. et ft. chart. vi. Sig. One t. i. d.

WHOOPING COUGH.

℞ Syr. ipecac..... ℥ iiss.
Syr. scillæ ..... ℥ iij.
Mist. assafœt.....ad ℥ ij.

M. Sig. ℥ i.-ij. every 3 hours.

Or,

℞ Zinci oxidi. ..... ℥ i.
Ext. bella..... gr. v.

M. et ft. pil. xxi. Sig. One t. i. d.

Or,

℞ Ac. nit. dil..... ℥ iij.
Tr. cardamom. co..... ℥ i.
Syr. simp..... ℥ iij.
Syr. cinnam..... ℥ vi.

M. Sig. ℥ ij. every 3 hours, and let them drink freely of:

℞ Phos. acid..... ℥ i.
Aq..... O.i

Or,

℞ Pot. brom..... ℥ ij.
Hydrat. chloral..... ℥ ss.
Ext. bella..... ℥ i.
Syr. tolu..... ℥ ij.

M. Sig. ℥ i. every half-hour till relieved.

Or,

℞ Camph. monobrom.....gr. v.
Syr. tolu..... ℥ i.
Mucild. acac..... q. s.

M. Sig. ℥ i. as required.

Or (If Severe).

℞ Fl. ext. hyos..... ℥ ss.
Aq. aurant..... ℥ iv.

M. Sig. ℥ i.-iv. every 3 hours.

DISINFECTANTS.

For Privies:—

Dissolve a pound of *sulphate of iron* in a gallon of water; or mix thoroughly in water the same quantity of *chloride of lime.*

For Water-closets, Bed-pans, etc.:—

Use *Labarraque's solution of chlorinated soda,* a fluid ounce to a quart of water; or *permanganate of potassium* (the crude permanganate is much cheaper than the crystallized and will answer equally as well), ten grains to a quart; or *carbolic acid,* twenty grains to the pint or quart. *Tar* is a very good disinfectant.

For Drinking-water:—

After filtering, add sufficient *permanganate of potassium* to make it of a pinkish color in a strong light.

For Articles of Clothing, etc.:—

If much soiled from the discharges of patients, they should be burned or boiled thoroughly. Use a solution of *permanganate of potassium,* one ounce to three gallons of water. Expose for several hours to a dry heat of from 200° to 250° F., all woollen articles and bedding which cannot be washed.

For Occupied Rooms and Houses:—

Ventilate freely and diffuse by spray through the air *Ledoyen's liquid solution of nitrate of lead;* or solid *chloride of lime* may be placed in shallow vessels, or sprinkle one-per-cent solution of *carbolic acid.* Whitewash the cellars. *Charcoal* and *quicklime,* especially charcoal, absorb gas, and thus aid in purifying the air.

For Hospital Wards:—

Disinfect, ventilate, and cleanse well with *Ledoyen's liquid,* or with *chloride of lime,* or *bromine* may be left exposed to the air in shallow vessels, or *iodine* moderately heated.

For Heaps of Filth, Solid or Semi-liquid:—

Cover with *charcoal* two or three inches deep, or with dry earth.

For Drains, Ditches, and Sewers:—

Disinfect with *sulphate of iron, coal-tar,* or *chloride of lime.* A pound of good chloride of lime will be sufficient for a 1,000 gallons of running sewage.

POISONS AND THEIR ANTIDOTES.

NEUROTIC POISONS.

Poisons.	Antidotes.
Aconite.	After thoroughly emptying the stomach, diffusible stimuli must be used both inwardly and outwardly (especially alcohol).
Alcohol.	Stomach-pump ; emetics ; cold affusions ; counter irritants ; strong coffee ; artificial respiration ; electro-magnetism ; hypodermic injections of atropia. It is better to administer *ammonia* as it is the *physiological antidote.*
Atropia and Belladonna.	Stomach-pump ; emetics (especially sulphate of copper in ten grain doses) ; cathartics ; cold affusions ; hypodermic injections of a morphia salt; magnetism; stimulants.
Calabar Bean (physostigma.)	Stomach-pump ; emetics ; cold affusions; counter irritation ; strong coffee ; hypodermic injections of atropia ; artificial respiration ; magnetism.
Cannabis Indica.	Stomach-pump ; strychnia (hypodermically) ; vegetable acids ; vinegar ; hot-spirits and water.
Chloral.	Same as Calabar Bean. Do not use artificial respiration. *Physiological antidote* is *strychnia.*
Conium.	Same as Aconite.
Digitalis.	Same as Aconite.
Dulcamarra.	Same as Belladonna.

Hydrocyanic Acid and Cyanides	Ammonia; chlorine; cold affusions; fresh air and artificial respiration.
Hyoscyamus.	Same as Belladonna.
Lobelia.	Same as Aconite.
Oil of Bitter Almonds.	Same as Hydrocyanic Acid.
Opium or Morphia.	Same as Calabar Bean.
Stramonium,	Same as Belladonna.
Strychnia.	Tannic acid; opium; conium; extract of hemp; camphor; chloral; calabar bean; bromide of potassium; inhalations of ether or chloroform; atropia.
Tobacco.	Same as Aconite.
Veratria.	Same as Strychnia.
Veratrum Viride	Same as Aconite.

CORROSIVE POISONS

Poisons.	Antidotes.
Acids (mineral and vegetable).	Magnesia; chalk; alkaline solutions; fixed oils; carbonate of soda. Do not use emetics.
Alkalies.	Citric acid; lemon juice; vinegar; oils.
Alum.	Carbonates of ammonium or sodium.
Arsenious Acid.	Hydrated magnesia; hydrated sesquioxide of iron (℥ i doses).
Baryta and its soluble salts.	Sulphates of magnesium, sodium, or potassium.
Bichromate of Potassium.	Alkaline carbonates; soap; magnesia.
Chromium compounds.	Chalk or magnesia compounds; emetics.

Corrosive sublimate and soluble mercurial salts.	White of egg ; milk ; flour ;—for cupric salts also ferrocyanide of potassium.
Cream of Tartar.	Sodium carbonates in solution.
Nitrate of Silver.	Chloride of sodium.
Salt of Sorrel.	Salts of calcium.
Soluble Arsenites.	Ferric subacetate.
Soluble Cupric Salts.	Same as Corrosive, Sublimate and Soluble Mercurial Salts.
Soluble Lead Salts.	Alkaline or soluble earthly sulphates ; diluted sulphuric acid ; emetics (for after treatment milk and opium should be given).
Soluble Zinc Salts	Albumen ; magnesia ; sodium carbonates; emetics ; demulcents (warm).
Sulphates and Chloride of Iron.	Alkaline carbonates.
Tartar Emetic.	Tannic acid ; catechu or any vegetable acids.

IRRITANT POISONS.

POISONS,	ANTIDOTES.
Arsenic.	Salts of iron (dyalized iron) ; magnesia ; lime and astringents ; artificial repiration ; ammonia ; charcoal ; cold affuions.
Arsenic (in solution)	Freshly precipitated hydrated sesqui-oxide of iron.
Asphyxiating Gases.	Cold affusions ; artificial respiration · magnetism.
Bromine.	Ammonia.

Cantharides.	Emetics; opiates (which may be given either by mouth or rectum) and demulcents.
Carbolic Acid.	Substances of an albuminous, mucilaginous or oleaginous nature.
Chlorine Gas.	Cautious inhalation of ammonia.
Chlorine Water and solutions of Chlorinated lime.	Albumen given freely in the form of milk, eggs, flour, etc.
Chloroform.	Artificial respiration; cold affusions; galvanism.
Colchicum,	Tannic acid; emetics; demulcents. Should symptoms of coma come on administer ammonia, brandy, coffee.
Creosote.	Same as Carbolic Acid.
Croton Oil,	Sulphate of copper (in gr. x. doses), to produce emesis, which should be followed by mucilaginous drinks containing opium.
Drastic Cathartics.	Demulcents; opiates; stimulants.
Iodine.	Starch or flour-water; demulcents; emetics.
Paris Green (arseniate of copper).	Same as Arsenic.
Phosphorus.	Magnesia; oil of turpentine (old); emetics; purgatives.

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